"A STUDY ON CORD BLOOD NUCLEATED RBC COUNT- A MARKER OF FETAL ASPHYXIA"

Dissertation submitted to

THE TAMILNADU Dr.M.G.R MEDICAL UNIVERSITY

In partial fulfilment of the requirement
for the award of

M.S.DEGREE – OBSTETRICS & GYNECOLOGY

BRANCH - II

KILPAUK MEDICAL COLLEGE
KILPAUK, CHENNAI.

APRIL 2015

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY ON CORD BLOOD NUCLEATED RBC COUNT- A MARKER OF FETAL ASPHYXIA"** is the bonafide original work of Dr.G.Thenmozhi under the guidance of Dr.T.K.Shaanthy Gunasingh MD.,DGO., Professor and Head of the department of Obstetrics and Gynaecology KMCH,Chennai in partial fulfilment of the requirements for MS Obstetrics and Gynaecology branch II examination of the Tamilnadu Dr.MGR Medical university to be held in April 2015 .The period of Postgraduate study and training from July 2013 to July 2015.

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DECLARATION

I solemnly declare that this **"A STUDY ON CORDBLOOD NUCLEATED RBC COUNT- A MARKER OF FETAL ASPHYXIA"** was prepared by me at Government Kilpauk Medical College and hospital,Chennai, under the guidance of **Dr.T.K.ShaanthyGunasingh,M.D.,D.G.O.,**Professor and Head of the Department,Department of Obstetrics and Gynaecology,Government Kilpauk Medical College and hospital,Chennai.

This dissertation is submitted to the **Tamil Nadu Dr.M.G.R. Medical University,Chennai** in partial fulfillment of the University regulations for the award of the degree of **M.S Obstetrics and Gynaecology.**

Place:

Date: (Dr.G.Thenmozhi)

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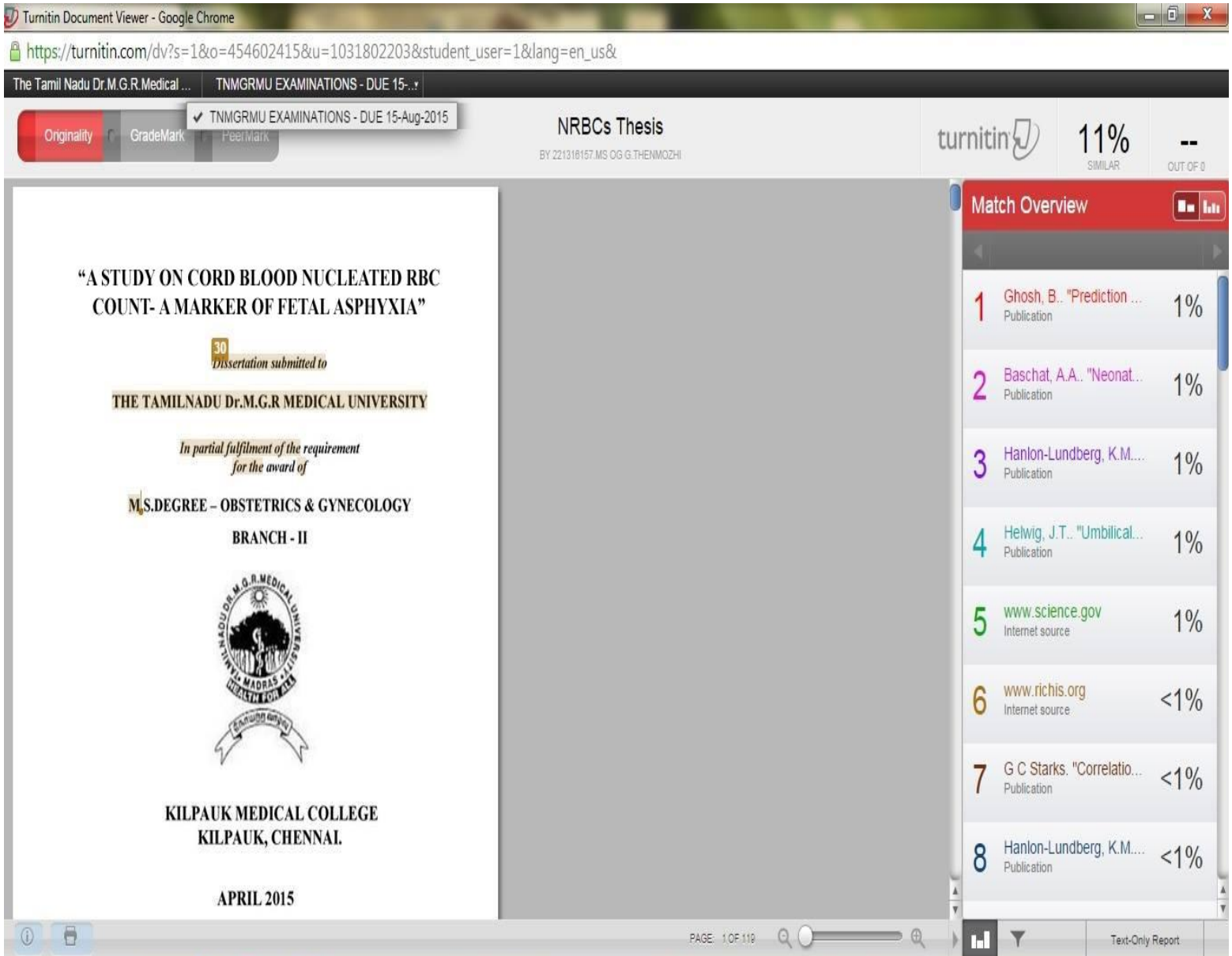
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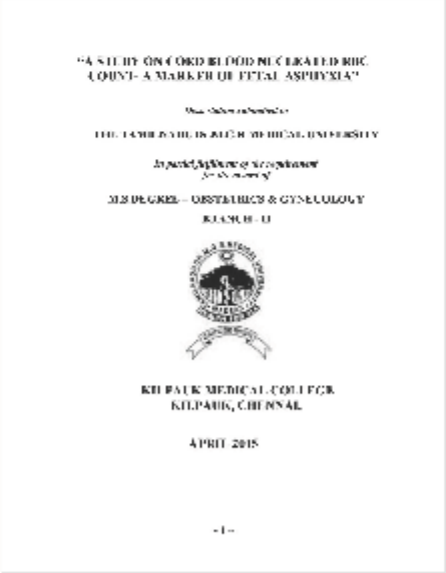

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ABSTRACT

BACKGROUND AND OBJECTIVES:

The objective of this study is an attempt to establish a relationship between the levels of nucleated RBC's and to assess the severity of perinatal asphyxia and early neonatal outcome. There by preventing complications such as hypoxic ischemic encephalopathy, neurological impairment and polycythemia.

METHODS:

In this study 320 patients who have undergone emergency LSCS at Govt kilpauk medical college were taken. Singleton term pregnancies primi /multi babies of more than 2.5kg appropriate for gestational age irrespective of indication, without any maternal co morbid factors were taken up.

Inclusion and exclusion criteria, study protocol were designed. Various parameters were also studied including NICU Admissions, relation with gravida, maternal age, LSCS indication, duration of labour.

RESULTS:

NRBC'S were significantly high in cord blood of patients with prolonged first and second stage of labour and who underwent emergency lscs for fetal distress and deep transverse arrest. Also increased NRBC'S were noted in babies

with low apgar score. Babies with birth asphyxia who were diagnosed by the pediatrician, showed an increased levels of NRBC'S. NRBC count increased proportionately to the severity of HIE.

CONCLUSION:

From this study it was concluded that estimating the number of nucleated RBC/100 WBC in umbilical cord venous blood sample of new born is an important test, the sample being obtained non invasively from otherwise discarded specimen and analyzed by personnel or equipment readily available in most hospital laboratories. The level of nucleated RBCs/100 WBCs correlates with acute intrapartum asphyxia and can be used as an index of early neonatal outcome.

CONTENTS

LIST OF ABBREVIATIONS USED

LSCS - Lower segment caesarian section

FTND - Full term normal vaginal delivery

Hb - Hemoglobin

HIE - Hypoxic ischemic encephalopathy

IUGR - Intra uterine growth restriction

FHR- Fetal heart rate

FD- Fetal distress

FI- Failed induction

DTA- Deep transverse arrest

MSL- Meconium stained liqour

AGA- Appropriate for gestational age

NICU - Neonatal Intensive care unit

NRBC - Nucleated Red Blood Cells

RBC - Red Blood Cells

SD - Standard deviation

UA - Umbilical artery

WBC - White Blood Cells

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INTRODUCTION

Science has allowed medicine to penetrate the hidden World of the fetus and to begin diagnosis and treat fetal conditions. To obstetricians, the fetus is "the patient within the patient", and part of the discipline of Obstetrics is the care of the fetus[13].

Fetal and neonatal death's leading cause world over is perinatal asphyxia. Perinatal asphyxia can be defined as clinical or biochemical evidence of decrease of oxygen and an increase of carbondioxide in the body because of the deficient respiratory function at birth with resultant hypoxia and acidemia[27]. In the developed countries the percentage of perinatal asphyxia is 2% as shown by LOW, 1998, but the overall percentage is around 5-10%. Cerebral palsy and mental retardation is reported in 8% cases following fetal asphyxia as shown by Blair and Stanley, 1988.

The obstetrician has a responsibility in recognizing the hypoxic event so that one can prevent associated morbidity and mortality[14.46]. Currently, many parameters are used to define or predict perinatal asphyxia.

Commonly used parameters are:

1. APGAR Score ,
2. Umbilical arterial acidemia/base excess,[37]
3. Presence of meconium in amniotic fluid ,

4. Intrapartum electronic fetal monitoring,
5. Nucleated red blood cells per 100 white blood cells in

   Umbilical cord (venous) blood smears.

NRBCs are commonly seen in the circulation of newborns. The number of NRBCs per 100 WBCs varies and it is usually less than 10. Conditions were there is >10 NRBC's are usually seen are prematurity, Rh sensitization, maternal diabetes mellitus and intra uterine growth retardation. Asphyxia is also said to cause an increase in the nucleated RBC'S in the newborns.[34]

Though perinatal asphyxia is commonly encountered there are no single variable that could assess the severity of the same, there are combination of various indices that could assess the degree of the severity are being used now.

The present study was done to evaluate the significance of presence of nucleated red blood cells/100 white blood cells in a blood smear made from umbilical cord blood (venous) sample. Therefore the aim of this study is to correlate the NRBC levels and acidemia in neonates.

The present study was carried out on 320 pregnant women admitted to the labour ward at Govt. kilpauk medical college and hospital, Chennai.

AIM AND OBJECTIVES OF THE STUDY:

1. To determine normal levels of nucleated red blood cells /100 white blood cells in cord blood smear of non asphyxiated term new borns.

2. To establish a relationship between the levels of nucleated red blood cells /100 white blood cells and to assess the severity of perinatal asphyxia .

3. To assess the short term neonatal outcome in asphyxiated babies.[perinatal period 1 wk]

4. To correlate NRBC's count with the neonatal outcome associated with perinatal asphyxia. [NICU admissions, HIE& its severity].

REVIEW OF LITERATURE:

Effect of Labour on the normal fetus

Uterine contractions are usually associated with reduced uteroplacental perfusion and decreased oxygenation of fetal blood even in normal labour[32]. When intrauterine pressure increases more than 30mmHg, uterine perfusion stops due to the increase in the intramyometrial pressure exceeding the arterial pressure. In the normal course of labour, with the fetus having normal oxygen reserve, the decrease in oxygen levels during contractions are usually above 17-18 mm hg which is the "critical level".[47]

The fetus normally can withstand stoppage of blood flow during uterine contractions occurring every 2-3 minutes without demonstrating abnormal fetal heart rate patterns or alterations in acid-base. However transient prolongation of the uterine contractions can lead to a "respiratory acidosis" due to diminished excretion of the fetal co2. When there is a decrease in the blood flow for a long time, it will impair severely the oxidative metabolisms even in the normal fetus. Under these circumstances, lactate and pyruvate are produced and accumulated by anaerobic glycolysis, which will lead to a metabolic acidosis[64].

Though the reserve capacity of "normal" pregnancies will be adequate to protect the fetus against the stress induced by "normal" labour, higher myometrial

activity in a “normal” pregnancy in abnormal situation may be sufficient to compromise the fetus[32].

Effect of Labour on the compromised fetus

During each uterine contraction with labour, there is a decrease or actual cessation of intervillous space blood flow, based on the uterine pressure, resulting in diminished respiratory gas exchange between the fetus and mother. Even when there is minimal stress to an already compromised fetus as in intrauterine growth retardation, it may cause a decrease of the fetal oxygen below tolerance levels and thereby causing hypoxic damage to the fetal central nervous system, which may even result in the death of the fetus[17]. “Normal” labour in a pregnancy which is complicated may be sufficient for the fetus to be compromised.

Effects of hypoxia on Fetus

Asphyxia is defined as insufficient exchange of respiratory gases[21]. This insufficiency is usually caused by inadequate umbilical or uterine blood flow, which results in a reduction of oxygen content and elevation of carbon dioxide in fetal blood. Eventually, it gives rise to physiologic compensatory mechanisms; the most important of this is redistribution of blood flow within the fetus. Blood flow for the heart, brain and adrenal gland increases; blood flow for the placenta is maintained; and blood flow decreases to all other areas [34].

There are many mechanisms causing asphyxia to the fetus during the stages of

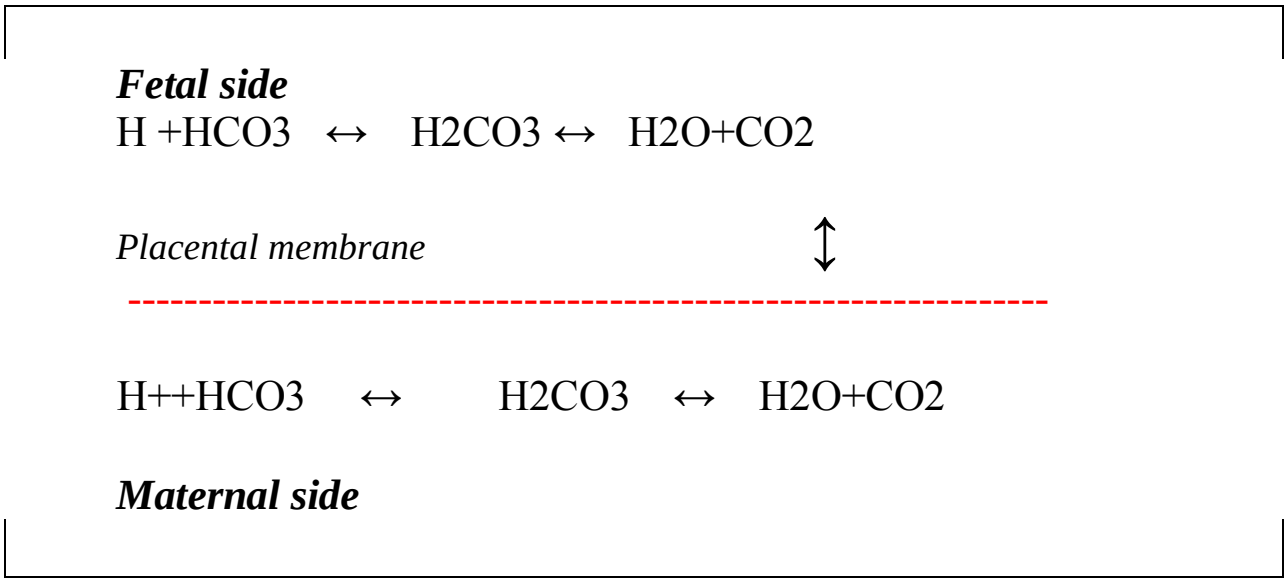
labor and to the neonate immediately after birth. Among them, few common causes are interruption of the umbilical circulation (cord compression), altered placental gas exchange (placental abruption , previa and insufficiency), inadequate perfusion of the maternal side of the placenta (maternal hypotension, hypertension from any cause or abnormal uterine contractions), impaired maternal oxygenation (cardiopulmonary disease, anemia) and failure of the neonate to accomplish lung inflation and successful transition from fetal to neonatal cardiopulmonary circulation[18].

CO2 exchange and respiratory acidosis

The fetal acid-base ratio depends on a bicarbonate buffer system though it is not as efficient inside the uterus as it occurs extrauterine, because the ability to eliminate carbon dioxide is effected mainly by the maternal respiration. There are many factors which affect fetal exchange there by affecting the ability to eliminate CO2. This condition is called as respiratory acidosis. The first biochemical change that occurs is an increase in fetal pCO2. The increase in pCO2 will causes an increase in fetal hydrogen (H+) ion concentration and thereby lowering of the pH. This happens due to the mechanism as shown in the figure 1. Any interference affecting the CO2 elimination will cause a shift of the bicarbonate buffer equation towards the left due to the formation of H+ ions. Similarly, any increased H+ ion production by the fetus, called as “metabolic acidosis”, which drives the equation

towards the right and causing an increase in pCO2.

Figure 1 Fetal bicarbonate buffer system.



The H+ ions produced in the intermediate metabolism at the fetus transformed into CO2. This CO2 is transferred by a pressure gradient into the maternal circulation and eventually eliminated by the maternal lungs.

O2 exchange and metabolic acidosis:

Decreased oxygen (O2) transfer is another important cause of acidosis in the fetus. For a sustain growth, development and normal pH balance a fetus requires 5 to 10 ml O2/kg /min. Decreased O2 supply to the fetus may occur suddenly as in the case of abruptio placentae, hypertonic labour etc. it can also occur as a chronic

process. When there is a acute and severe fetal hypoxia such as in abruptio placentae, there is very less time for adaptation to occur due the sudden decrease in PO2, signs and symptoms become firstly apparent. If hypoxia to the fetus is chronic, in cases such as the maternal chronic hypertension, the fetus will temporarily adapt. The decreased generation of adenosine triphosphate (ATP) due to chronic O2 deprivation will affect fetal growth, and the ability of the fetus to tolerate stressful situation will be seriously compromised[6].

Acute hypoxia usually presents with prolonged bradycardia < 80 bpm, while at the same time **sub acute hypoxia** presents with steep decelerations reducing the FHR to < 80 bpm and which last longer than the time the FHR is at the normal baseline rate.

The above two patterns can lead to acute clinical events such as placental abruption, cord prolapse or scar rupture. It do occur in the late first or second stage of labour. Many a times the cause is not known, may be related to occult cord compression.

Gradually developing hypoxia may be manifested as development of tachycardia, reduced variability and absence of accelerations. **Longstanding hypoxia** does show a pattern with reduced baseline variability and shallow late decelerations in a non-reactive trace.

Acute hypoxia:

Continuous bradycardia or deceleration < 80 bpm leads to acute hypoxia, if it is associated with placental abruption, cord prolapse and uterine scar rupture which warrants immediate delivery. Uterine hyperstimulation causing bradycardia can be acted upon by acute tocolysis.

Other important considerations in cases are the cardiotocograph (CTG) prior to the bradycardia and to potentially assess associations such as thick meconium stained amniotic fluid, infection, intrauterine growth restriction (IUGR) and antepartum hemorrhage in which acidosis can occur rapidly.

A FHR < 80 bpm for longer than 6 minutes, prolonged bradycardia/prolonged deceleration, can lead to acidosis and hypoxia. A prolonged deceleration <3 minutes is considered suspicious and > 3 minutes is abnormal. Causes of transient bradycardia include dorsal position of the mother, hypotension induced by regional anaesthesia, artificial rupture of the membranes, uterine hyperstimulation and vaginal examination. In these cases quick actions should be undertaken, such as maternal repositioning, correction of hypotension, stopping oxytocin and acute tocolysis for hyperstimulation with prostaglandins whilst awaiting recovery of the fetal heart rate[41].

While crowning, the pressure on the head with maternal bearing down in the second stage may also be associated with bradycardia. If it fails to recover within 6

minutes, delivery should be facilitated. At times the cause for bradycardia is not known and the fetal heart rate may not recover, despite the usual resuscitative measures, necessitating immediate delivery. The longer the duration of bradycardia the greater is the chance for fetal acidosis[23]. The pH is more likely to decline rapidly in high-risk clinical situations such as the oligohydramnios, thick meconium, intrauterine infection, IUGR, and in cases where the CTG was suspicious or pathological before the onset of bradycardia. In these cases if the bradycardia fails to recover by 6 to 7 minutes delivery should be undertaken as soon as possible.

The placenta acts as the lungs in utero for the fetus. Carbon dioxide is eliminated at the same time oxygen is absorbed through the placenta[45]. For optimal gas exchange to take place there should be adequate circulation occurring between the maternal and fetal sides of the placenta. With the normal fetal heart rate (FHR) of about 150 bpm for 10 minutes there are 1500 circulations that occur through the placenta which will help transfer carbon dioxide out of the fetal circulation and at the same time absorb adequate oxygen for the fetus. When the FHR is 80 bpm there will be only 800 circulations in same period of time that is 10 minutes the fetus will miss 700 circulations[67]. The total amount of carbon dioxide excreted becomes less which accumulates within the fetus, leading to the formation of carbonic acid causing a decline in pH thereby causing “respiratory acidosis”. With

increase in the duration of bradycardia the oxygen delivered to the fetus is also reduced, leading to anaerobic metabolism and thereby causing accumulation of metabolizes. The above mentioned phenomenon gives rise to metabolic acidosis. This along with the already existing respiratory acidosis acts as a additive effect. If the fetal heart rate returns back to normal within a short period of time the number of circulations occurring through the placenta will normalize there by allowing the respiratory acidosis to be corrected by it, which occurs by transferring the carbon dioxide to the maternal circulation. This is a fast process while reversal of metabolic acidosis takes longer time. If these conservative measures fail, and if the fetal heart rate does not return to normal within 6-9 minutes, delivery of the fetus and establishing neonatal respiration quickly will reverse the respiratory acidosis and with time the metabolic acidosis[80].

Subacute hypoxia

Prolonged decelerations occurring in the FHR below the baseline for a long time than occurring at the normal baseline rate leads to subacute hypoxia, i.e. the development of hypoxia and acidosis, but less quickly compared with acute and prolonged bradycardia. When such fetal heart rate decelerations are frequent and profound, the evolution of hypoxia and acidosis can be fast. It is difficult to quantify the exact duration for which the FHR should be below the baseline rate and the duration of time for which it should be at the correct baseline to prevent

acidosis and hypoxia. It will depend on the 'physiological reserve' of each fetus. One should consider that the buildup of acidosis and hypoxia is likely to be greater if the duration of the FHR at the normal baseline rate is one-third or less of the total time[36]. Initially this will cause a slow elimination of carbon dioxide there by leading to respiratory acidosis, but as time passes by the oxygen transfer will be critically reduced and metabolic acidosis will start to develop.

Gradually developing hypoxia:

In gradually developing hypoxia decelerations do occur which is followed by absence of accelerations, a rise in the baseline rate, and reduction in baseline variability. As always one should consider the clinical picture of cervical dilatation, parity, rate of progress and high-risk factors. Start instituting conservative measures such as stopping oxytocin, hydration, and change of maternal positions or consider delivery. Decelerations progressively become more pronounced, accelerations disappear there is a rise in baseline rate and finally a reduction of the baseline variability. The decelerations are variable and are suggestive of cord compression[55].

Longstanding hypoxia:

In cases with longstanding hypoxia there is no accelerations, the baseline variability is significantly reduced and there will be shallow late decelerations,

often < 15 bpm. These characteristic features of hypoxia are seen even though there is a normal baseline rate in the fetus. The absence of accelerations and 'cycling' suggests that the fetus could have already had sustained asphyxial injury, or is hypoxic, or is affected by few other insult such as infection[28].

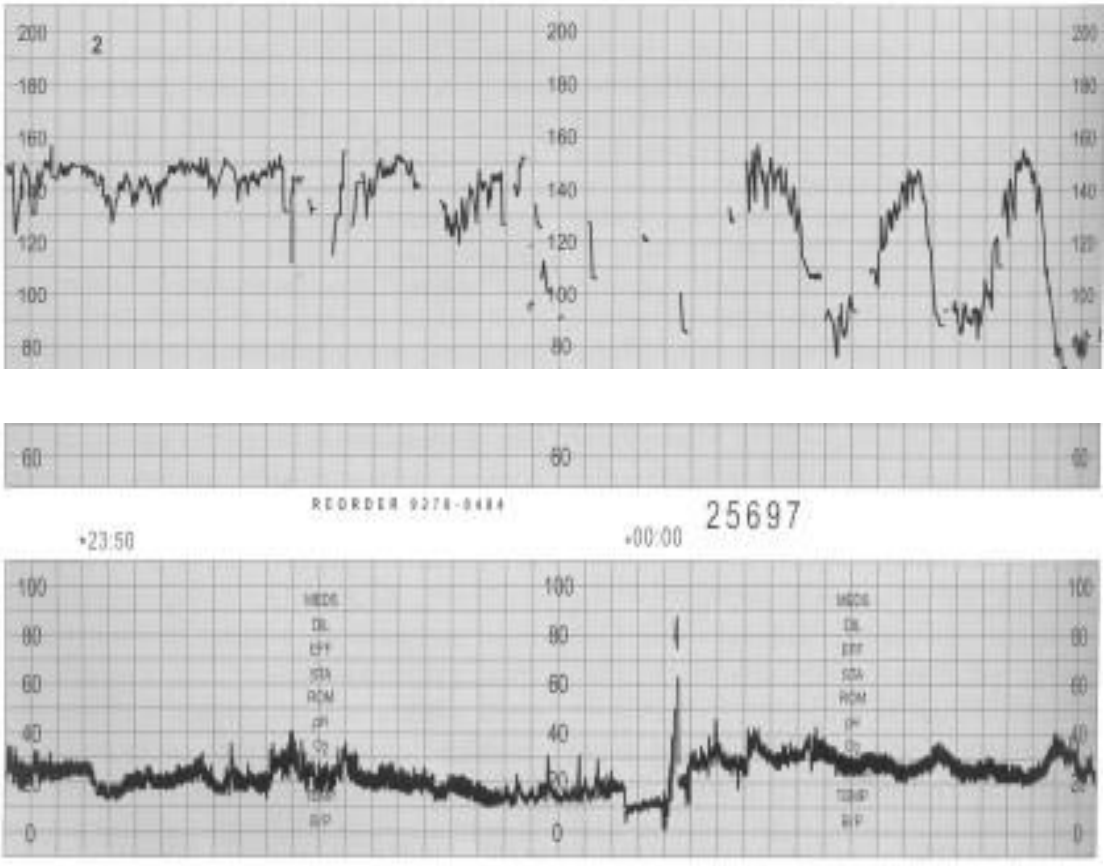


Figure 2.Decelerations start as shallow and then get steeper and wider lasting for 2 minutes and recovering to the baseline rate of 140 bpm

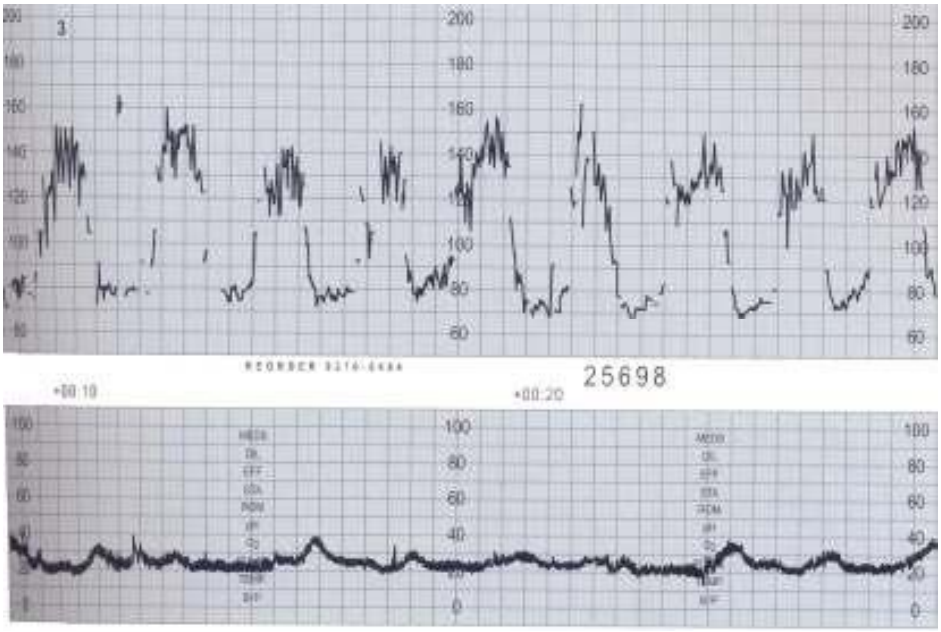


Figure 3: prolonged decelerations with salutatory baseline variability during short period of recovery

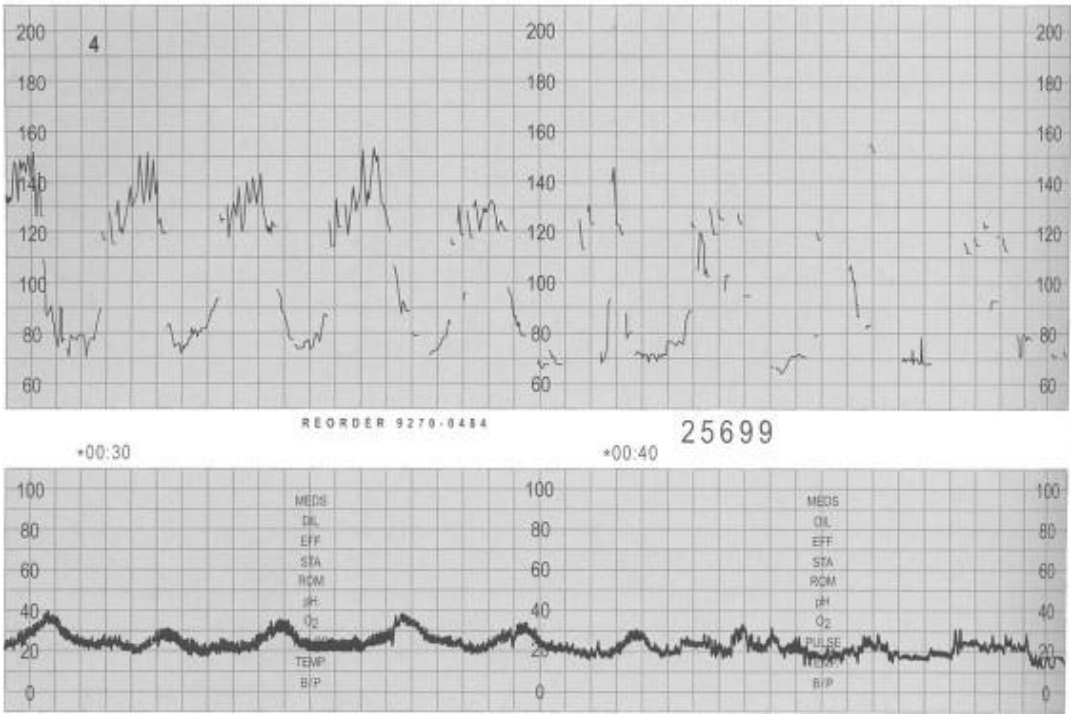


Figure 4:The baseline rate drops from 150 bpm to 120 bpm with prolonged

decelerations.

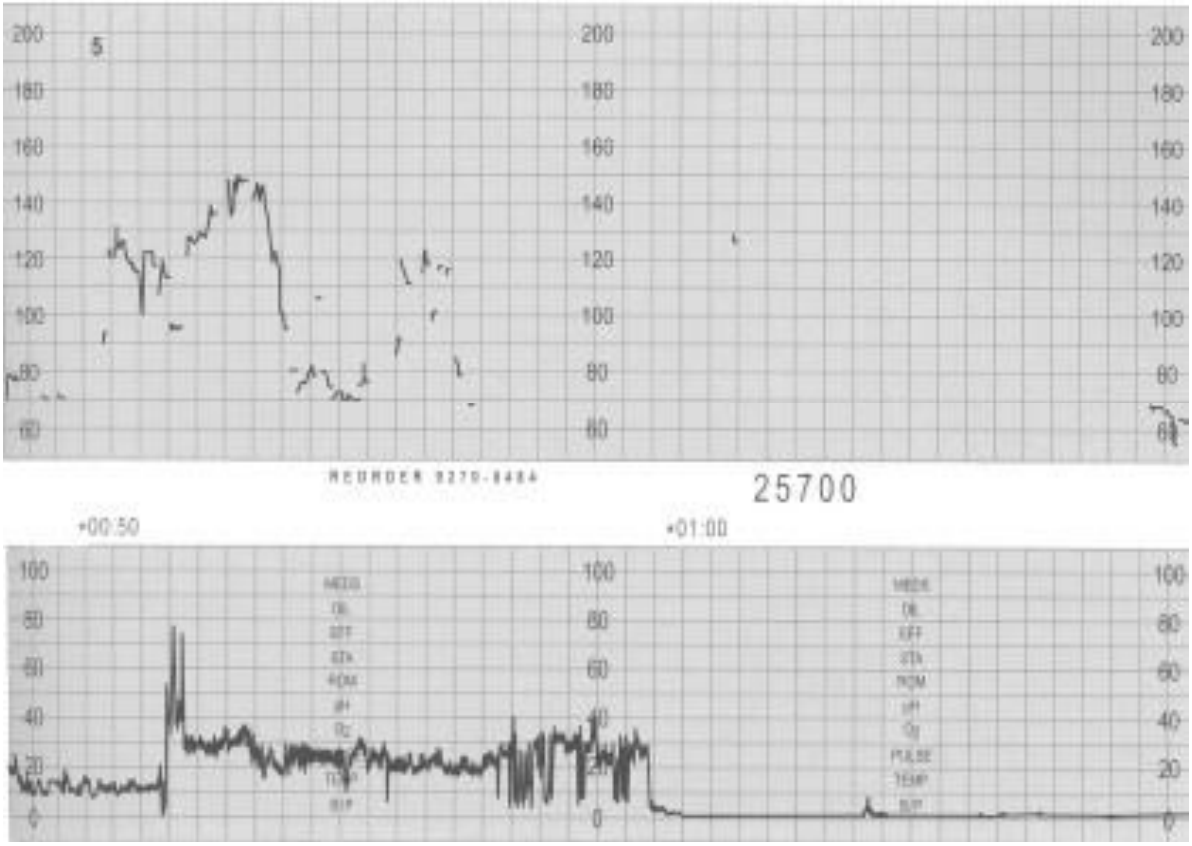


Figure5: Prolonged bradycardia following decelerations

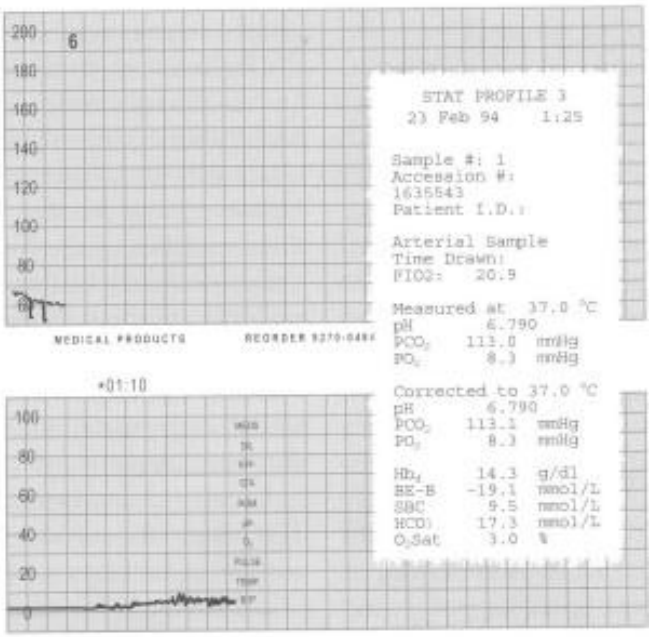


Figure 6: Time of delivery is annotated and the strip shows a low and high base excess.

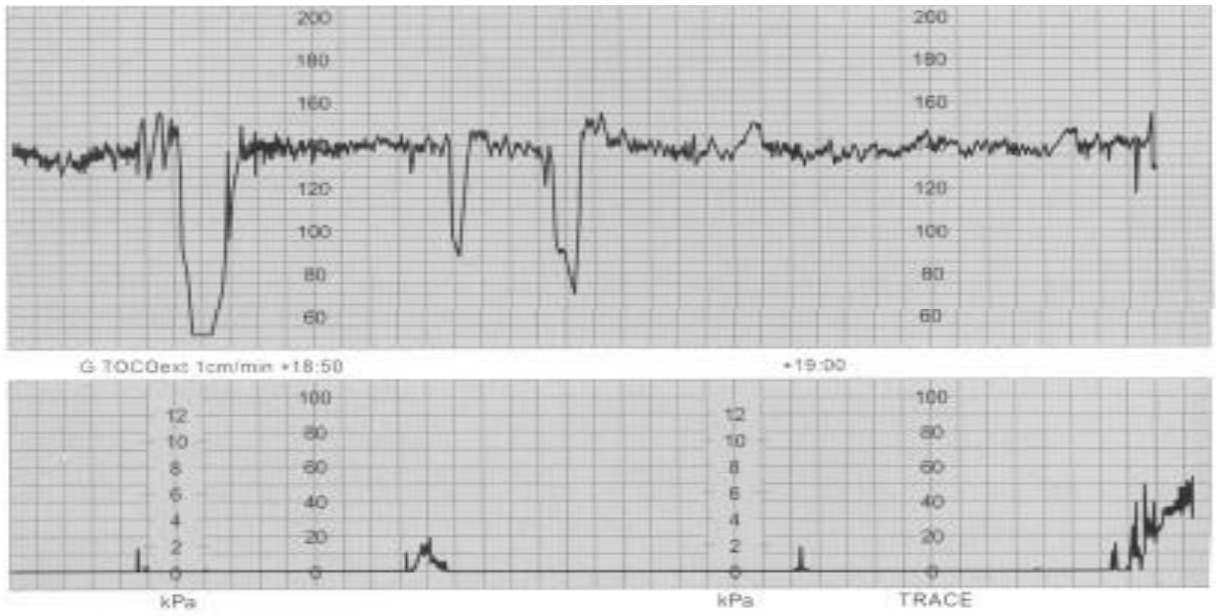


Figure 7: *The trace shows a baseline rate of 140 bpm with simple variable decelerations, normal baseline variability and accelerations.*

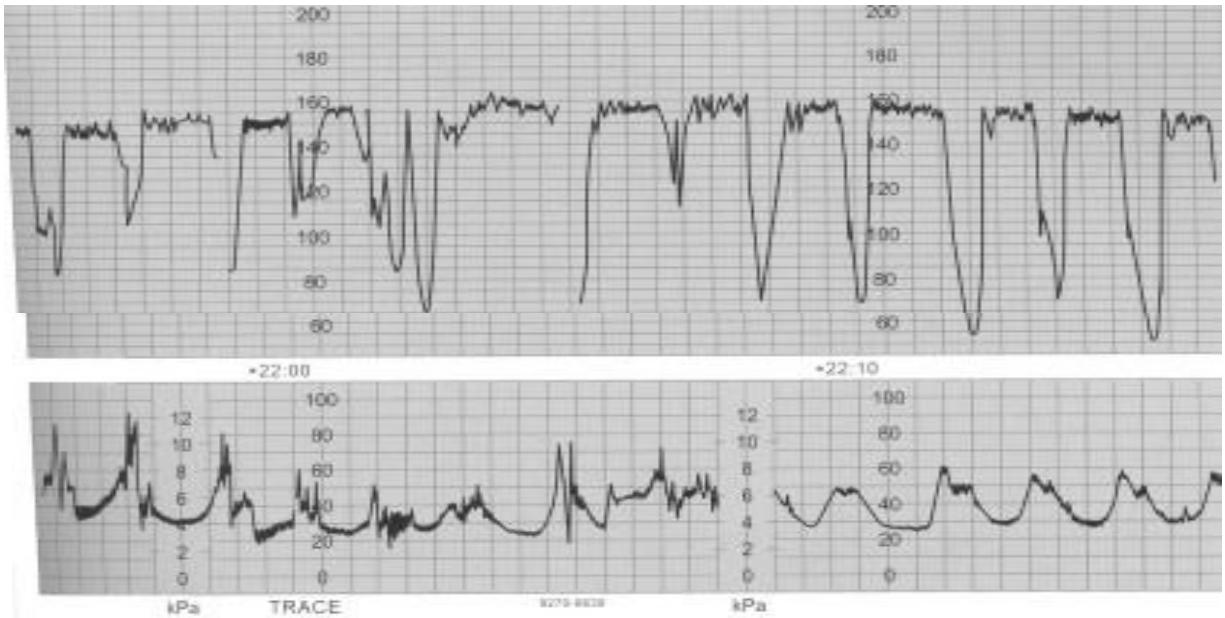


Figure 8: *Rise in baseline rate to 150 bpm, reduced baseline variability and no accelerations.*

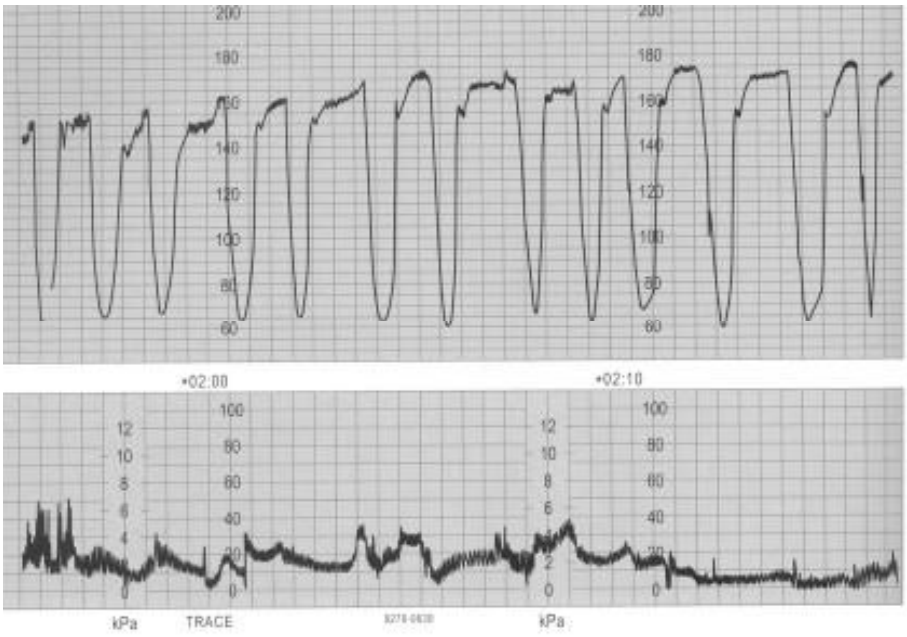


Figure 9: *Rise in the baseline rate to 170 bpm with no baseline variability.*

Commonly used clinical parameters used in the diagnosis of fetal asphyxia are analyzed below:

- Apgar score
- Umbilical Cord blood acidemia/ base excess
- Intra partum fetal monitoring
- Presence of meconium in amniotic fluid

1) Apgar score:

Introduced by Virginia Apgar in the year 1953, the Apgar score is taken as a quick (although subjective) measure of the outcome of labour and delivery including expected status of a neonate at birth.

It is comprised of five components -

Heart rate,

Respiratory effort,

Muscle Tone,

Reflex irritability,

Colour,

Each of which will be given a score of 0, 1 or 2. At one minute a score of 0 to 3 will indicate severe birth asphyxia, a score of 4 to 5 do indicate moderate birth asphyxia, requiring active intervention by means of resuscitation (Nelson et al 1996). However, its use has been associated with a lot of fallacies. The American Academy of Pediatrics in the year 1986 has challenged the use of Apgar score alone to define birth asphyxia. The committee came to a conclusion that although low Apgar score at 1 and 5 minutes does identify infants who need resuscitation. The Apgar score alone is not an evidence of sufficient hypoxia to result in neurological damage.

Stykes et al (1982) in a prospective study of 1210 deliveries studied the

relationship between Apgar score and Acid based status at birth[37]. Only 21% of babies with Apgar score at one minute of less than seven and 19% of babies with Apgar score at 5 minutes of less than 7 had severe acidemia (pH<. 7.1). On the other hand, 73% of babies with severe acidosis had Apgar score at one minute of >7 and 86% had an Apgar score of >7 at 5 minutes. The study questioned the value of Apgar score as an Index for assessment of asphyxia. Silverman et al (1985) reported poor correlation of Apgar score with bio chemical abnormalities in Cord blood. In those new borns with severe hypoxemia (pO2 < 10) 87.9% had a 5 minutes Apgar score of > 7. Similarly, 69.1% of new borns with pCo2 > 65 had an Apgar score of > 7 at 5 minutes[20].

Nelson and Jonas (1981) attempted to evaluate the utility of Apgar score in the prediction of long term neurological morbidity. When followed upto the age of 7 years, 55% of children with cerebral palsy had an Apgar score of 7 to 10 at 1 minute. In those with prolonged low Apgar score i.e. scores of 0 to 3 at 10, 15 or 20minutes, 12% developed cerebral palsy whereas 8% were free of major handicaps at early school age. The authors concluded that the utility of taking the Apgar score as an Index of Prediction of neurological development is questionable[11].

2) Umbilical cord blood acid base analysis

It is commonly believed that determination of fetal cord blood acid base status at delivery is a sensitive and objective mean of assessing the immediate new born condition and intra partum events (Low 1988). However, considerable controversy exists about optimal cut off value to diagnose intra partum asphyxia.

Yeomans et al (1985) evaluated cord blood from 146 infants born after uncomplicated vaginal deliveries between 37 and 42 weeks period of gestation. The infants born to women with pregnancy complications (diabetes Mellitus/ IUGR/ Meconium stained liquor/ pregnancy induced hypertension/twins) were excluded. The mean (+ SD) umbilical arterial blood gas values were as follows: pH 7.28(± 0.05), pCo2 49.2 (±_8.4), p02 (±_6.2), mm of Hg. Thus the lower limit of normal pH (mean - 2SD) was 7.18[81].

Various reported series have mentioned different normal blood gas values with a cut off between normal and acidemic infants ranging from less than 7.11(Stykes et al 1982) to less than 7.2 (Vintzileos et al 1987)

Table 1*: Normal values of Umb cord acid-base parameter in various studies*

AUTHOR	**UA pH (Mean SD)**	**UA pO2 (Mean SD)**	**UA pCo2 (Mean SD)**
Eskaetal, 1983	7.23(0.07)	X	x
Yeoman et al, 1985	7.28(0.05)	18(6)	49(8)
Low 1988	7.262(0.07)	15.2(4.9)	44.9(9.9)
Thorp et al, 1989	7.24(0.07)	18(7)	56(9)

UA-Umbilical artery

The ACOG committee opinion (1994) states that fetal acidemia is defined as Umbilical cord, Arterial blood pH of less than 7.2 denotes an arbitrarily high level. In normal uncomplicated pregnancies a lower normal range of 7.1 to 7.15 has been reported. The precise cut off value to define acidemia is unknown, but values less than 7.0 represent clinical significant acidosis. However, the presence of acidemia by itself is not sufficient to establish that a hypoxic injury has occurred[2].

Yoon & Kim (1994) conducted a trial of 356 singleton term new borns to determine the effects of labour and duration of second stage of labour of U. Cord blood acid base status [82](Table 2 & 3). Patients were divided into 3 groups.

A) Those who underwent caesarean section in absence of labour (n= 135)

B) Those who underwent caesarean section in first stage of labour (n = 62).

C) Those who had vaginal deliveries (n = 159).

Table 2: Effect of mode of delivery on Umbilical Artery Acid base status (Yoon & Kim 1994)

UA Acid base status	**Group A (n = 135)**	**Group B (n = 62)**	**Group C (n = 159)**	**p value**
pH (mean ± SD)	7.27 ± 0.05	7.26 ± 0.05	7.24 ± 0.07	< 0.05
PCO2 (mean ± SD)	57.3 ± 7.1	50.5 ± 7.5	49.4 ± 10.5	NS
PO2 (mean ± SD)	23.4 ± 8.9	22.1 ± 7.9	22.7 ± 8.3	NS

UA indicates Umbilical Artery

Table 3: Effect of duration of second stage of labour on U.A. Acid basis status (Yoon & Kim 1994)

Acid base status	**Duration of second stage (minutes)**			
	1 – 30	**31 – 60**	**> 60**	**p value**
pH (mean ± SD)	7.25 ± 0.07	7.22 ± 0.06	7.20 ± 0.072	< 0.05
PCO2 (mean ± SD)	49.0 ± 9.4	48.5 ± 10.8	57.4 ± 15.4	NS
PO2 (mean ± SD)	22.8 ± 8.3	22.0 ± 8.0	27.4 ± 9.0	NS

The authors concluded that there is a significant fall in pH in the presence of

labour and increased duration of second stage of labour which should be taken into consideration in evaluating neo natal well being by cord blood pH measurements.

In a contradictory study, Wood et al (1973) found no significant acid based change related to total duration of second stage of labour unless specific delay in delivery occurred.

Various studies have also studied the effect of timing of clamping of cord and sample storage on the different acid based parameters.

Lievaart and dejong (1984) conducted a study in which they showed that with delayed clamping of cord after birth (more than 30 second interval) there was a significant fall in pH and rise of PCO2 values. This was so only for arterial blood and not for venous blood. He thus concluded that immediate clamping seems mandatory if acid base equilibrium of cord arterial blood is used to judge neonatal status[56].

The timing of sample collection is also known to influence the acid base status. Umbilical arterial blood of 25 patients were sampled from clamped umbilical cord segments every 15 minute for one hour after delivery (Duerback et al 1992). The samples were collected in non-heparinized, non-iced, plastic syringes and processed immediately after sampling. The initial and final lab values were noted.

Table 4: Effect of timing of sample collection in UA acid base status (Duerback et al 1992)

UA Acid base status	**0 minutes**	**60 minutes**	**p value**
pH (mean ± SD)	7.271 ± 0.04	7.267 ± 0.05	< 0.05
PCO2 (mean ± SD)	48.24 ± 6.96	46.65 ± 8.18	NS
PO2 (mean ± SD)	17.04 ± 2.76	17.0 ± 4.07	NS

The results showed no significant difference in pH, PCO2 and PO2 values in umbilical arterial blood for upto 1 hour of delivery.

In another study to demonstrate any change in pH, PCO2 and PO2 values at 10 minutes interval over a total of 30 minutes when samples were stored in syringes or clamped for segments, Owen et al (1995) concluded that small but significant changes were seen in acid based parameters in both groups over time. Storage in syringes, analysis showed no significant differences in pH and base deficit values whereas storage in clamped cord segments demonstrated no change in PO2 and PCO2 over a period of 30 minutes.

The importance of acid base status as a potential bench mark for neonatal outcome in pre term deliveries is demonstrated by following two trials:

Dickinson et al (1992) reviewed the umbilical arterial and Venus blood pH,

PCO2 and PO2 values in 1872 infants born between 24-36 weeks of gestation .

Table 5: Normal means Umbilical artery acid base status in pre term infants (Dickinson et al (1992)

UA Acid base status	**Umbilical Artery**	**Umbilical vein**
pH (mean ± SD)	7.26 ± 0.08	7.33 ± 0.07
PCO2 (mean ± SD)	53.0 ± 10.0	43.4 ± 8.3
PO2 (mean ± SD)	19.0 ± 7.9	29.2 ± 9.7

Correlation with Apgar score was poor and 82% of neonates with 5 minutes Apgar score less than 7 had normal blood gas analysis. There was also no significant difference in acid base status at birth between pre term infant and 1924 term infants who delivered within the same time period at the hospital.

Table 6: *Umbilical artery acid base status in pre-term and term new barns (Dickinson et al (1992)*

UA Acid base status	Pre-term	Term
pH (mean ± SD)	7.26 ± 0.07	7.242 ± 0.07
PCO2 (mean ± SD)	53.8 ± 10.4	56.4 ± 8.6
PO2 (mean ± SD)	18.4 ± 6.7	17.9 ± 8.9

The Apgar scores are generally lower in otherwise uncomplicated preterm newborns than in term newborns. This is of questionable significance. Umbilical

artery acid base analysis provides an objective assessment of newborns.

Ranin et al (1989) included in their study 77 otherwise uncomplicated preterms infants and 1292 uncomplicated term infants. The pre-term infants had significantly lower 1 minute and 5 minute Apgar scores, but there was no significant difference in the frequency of acidemia. The mean pH was 7.29 in pre-term group and 7.28 in the term group. The values of pH, pCO2, PO2 and HCO3 and base deficit were also similar in two groups.

Nagel et al (1995) performed neuro developmental assessment in children born with severe umbilical artery acidemia (pH <7). Out of 30 patients pH >7.0, 23 were admitted to NICU, 8 of whom required incubation. 28 children survived the neonatal period out of whom only one had mild developmental delay, when followed upto 3 years of age.

This goes to show that although academia is associated with considerable short term morbidity, those who leave the neo natal intensive care unit without major problem have good outcome.

3. Intrapartum Assessment

Fetal scalp blood studies

The addition of fetal scalp capillary blood sampling to fetal heart rate monitoring for Intrapartum surveillance was expected to reduce the false-positive rate in the diagnosis of fetal distress. However, it soon became apparent that false-

positive and false-negative results also occurred frequently in this technique. Approximately 37% of depressed neonates with 1-minute Apgar score less than seven had normal scalp pH and 30% of acidotic babies had normal Apgar score in one study. In addition, the relationship of abnormal fetal heart rate patterns to acidosis is not well defined, and discrepancies could not be explained by delays in collection or the assay of fetal scalp blood samples. Fetal scalp blood, connective tissue and umbilical artery pH all were shown to correlate in studies using a miniature electrode, to measure fetal scalp tissue pH continuously during labor. They did not correspond exactly however tissue pH usually was 0.04 units less than blood pH, which is thought to be physiologic. Various fetal heart rate patterns were correlated with pH changes: there was a decline in pH in fetuses with reduced variability of the base line heart rate in 36% of cases with tachycardia in 60% with variable decelerations. With late recovery and late decelerations in 93%.The pathophysiology of abnormal fetal heart rates explained the many false-positive results because the change in pH was related to the degree and duration of fetal stress and the fetal buffering capacity. Conversely, fetal heart rate patterns were a result of multiple factors mediated by fetal autonomic nervous system.

Nonreassuring fetal heart rate patterns should be evaluated to manage labor properly, preventing unnecessary Caesarean deliveries while ensuring fetal health. Fetal scalp blood and umbilical blood pH are the best adjuncts available currently.

Fetal scalp blood analysis improves diagnostic accuracy of fetal heart rate interpretations. Umbilical blood analysis helps to confirm the diagnosis in the neonate. The maternal pH and placental acid- base balance are important contributors.

To the fetal blood pH thus different aspects of the fetal condition are addressed by these different parameters, explaining the difficulty in using them clinically and their frequent correlation in extremes of fetal decomposition.

Attempts to increase the accuracy of the diagnosis by means of multiple scalp electrodes to measure pO2, pCO2 and pH have not improved. On the precision of diagnosis, they have not been tried in large scale control studies. Currently, none of the continuous electrodes have been used successfully outside clinical research centers although their correlation with umbilical artery blood samples is good.

SCALP STIMULATION TEST

An alternative to fetal scalp sampling is the scalp stimulation test. Based on the reactivity of the autonomic nervous system in a well compensated fetus, this test shares the problems of limited number of samples with abnormal umbilical blood values, and insufficient data to comment on both the false-negative false–positive rates. However there is a good correlation between fetal heart rate accelerations and normal scalp blood pH

VIBROACOUSTIC STIMULATION

Similarly, the addition of vibroacoustic stimulation to evaluate the fetus during labour correlates the reactive fetus with normal pH levels. In one study, there were 12 patients without fetal heart rate variability and 33% of these responded to this test; 11 had tachycardia and 55% responded; and seven had repetitive late decelerations and 0% responded. Of the 34 non responders of the total of 64 tested, 18 had a pH less than 7.25. There were 5 who reacted to scalp but not to vibroacoustic stimulation and two of these had a pH of 7.25 or less. Another study concurred that vibroacoustic stimulation was more likely to induce an acceleration than fetal scalp stimulation in the non acidotic fetus however a 24 pH below 7.25 was found in 43.7 % of fetuses who responded to this test, but there was no response in those with a pH less than 7.20. Much larger series are needed to evaluate the validity of these tests.

FETAL ELECTROCARDIOGRAPHY

Fetal electrocardiographic changes in labour have been used to evaluate the intrapartum fetus for acidemia and low Apgar score; some studies show a correlation of upto 99.3%of fetuses with T/QRS ratios less than 0.25 and normal pH values in scalp and umbilical blood. However other studies found no T/QRS ratio association with scalp blood or umbilical artery pH. Additional research is

needed to determine the role of fetal electrocardiographic changes in the diagnosis of fetal academia.

BIOPHYSICAL PROFILE

Intrapartum application of the biophysical profile has not been useful to identify the fetus with acidemia. In one study; only a nonreactive non stress test had any association with low pH and metabolic acidemia; 5 of 10 infants with a pH of 7.20 or less had biophysical profile scores of 8 or more. The biophysical profile appears to be unreliable in Labour.

This mode of intra partum surveillance came into vogue in 1960s (Connigham et al 1997) and has been widely used to identify the periods of fetal risks with the hope that clinical intervention would avoid the potential for perinatal death or neurological damage. The major impediment to progress in evaluation and investigation of fetal heart rate monitoring is lack of agreement in definitions and nomenclature of fetal heart rate pattern inspite of being in clinical use for three decades now. The National Institute of Child Health and Human Development (NICHD) Research Planning workshop in 1997 laid down standardized and unambiguous definition for fetal heart rate tracings in terms of base line fetal heart rate, base line variability acceleration, late acceleration, early deceleration, variable deceleration and prolonged deceleration. However, a consensus was not reached in the workshop regarding strict guidelines for clinical management using fetal heart

rate patterns.

Vintzileos et al (1995) compared the use of continuous electronic fetal monitoring with intermittent auscultation in improving the pregnancy out come. The meta analysis of a total of 18561 patients showed that the patients monitored electronically had significantly higher caesarean section rates (odd ratio = 1.23) and lower perinatal mortality due to fetal asphyxia (odd ratio = 0.41).

In a prospective randomized study of 504 patients Kelso et al (1978) compared electronic fetal monitoring with intermittent auscultation. The result showed that there was no significant difference between two groups in terms of neonatal deaths, apgar score, maternal and neo natal morbidity and cord blood gases. The caesarean section rate was significantly increased in the continuously monitored group but did not see attributable to fetal monitoring. Thus, no beneficial or deleterious effects of continuous fetal heart monitoring in labour was shown.

In a similar study in 1979, Haverkamp et al conducted a trial of electronic fetal heart rate monitoring versus intermittent auscultation in 690 patients. NO significant difference in immediate neonatal outcome (Apgar score, Cord blood gases, Neonatal morbidity and nursery course) was noted. The caesarean section rate was 18% First Group and 6% intermittent auscultation group. Thus the author concluded that electronic monitoring did not improve neonatal outcome. ON the

other hand mothers were at increased risk of caesarean section.

The neurological development of 50 children related to fetal heart rate pattern, intra partum was prospectively studied by Painter et al(1988). They noted a statistically significant difference in favour of children with normal fetal heart patterns in first year of life. However, when followed upto 6-9 years of age the difference in cognitive and neurological development was no longer evident, thus refuting the belief that brief abnormal fetal heart patterns during labour are indicative of irreversible central nervous system injury.

3) Meconium Stained Liquor

The appearance of meconium during any stage of labour has long been considered a clinical sign of fetal distress. Its incidence has been variously reported as 8-22% of all deliveries by different authors [Gregory et al (1974); Davies et al (1985), Low (1988)]

Composition of Meconium

- Water content – 72%
- Dry content – 28%
- Carbohydrate – 80%
- Protein and liquid – Minimal
- Nitrogen – High

• Bilirubin – 16mmol/gm

Pathogenesis of presence of Meconium in Liquor:

It has been hypothesized that any decrease in oxygen concentration of fetal blood induces hyperperistalsis of fetal gut and relaxation of anal sphincter with resultant passage of meconium (Barham, 1969). The hormonal control of meconium passage has been proposed to be based on the higher umbilical cord Motilin levels in those infants who have passed meconium, compared to those with clear liquor. This is also maturationally dependent with higher values in post term infants than pre-term infants (Lucas et al, 1979).

Etiology and pathophysiology of MSAF:

Meconium staining of amniotic fluid is found in 9-22% of all pregnancies, the passage of meconium as a sign of fetal distress was described by Shwartz in 1958. Walker (1954) found that meconium staining of amniotic fluid occurs when o2 saturation of umbilical venous blood falls below 30%, approximately one half of the normal at term. Fenton and Steer (1962) considered the passage of meconium in utero to be a normal physiological function of a term and post term fetus in which meconium staining is merely an indication of fetal maturity. Another explanation of fetal meconium passage in the absence of other signs of

fetal distress is by the compression of the umbilical cord which elicits a vagal response causing increased gastro intestinal motility, dilatation of the anal sphincter and meconium passage.

In 1968, Saling proposed that the fetal gut becomes ischemic as a result of mesenteric constriction due to hypoxia. This vasoconstriction represents part of the "diving reflex" seen in many vertebrates responding to hypoxia. As a result of this there is adequate blood supply and oxygen to vital structures like the heart and brain.

The blood supply to skin, kidney, and musculature of gut is decreased .Intestinal ischemia leads to transient hyperperistalsis, relaxation of anal sphincter and meconium passage in hypoxic fetuses.Thus the meconium staining of amniotic fluid is seen more frequently with decreased supply of oxygenated blood to the fetus which is seen in mothers with hypertension, anemia, chronic pulmonary disease or prolonged gestation.

Abramovici et al are of the opinion that the fetuses who have passed meconium during labour are in the state of temporary compensated fetal distress with well oxygenated vital organs and peripheral hypoxia.At this stage the fetal blood pH will show no acidemia and Apgar score will be good if delivered within reasonable period of time. A change in the fetal scalp pH would indicate decompensated fetal distress, in which intervention should be performed.

The passage of meconium is caused by one or a combination of the above causes. The mechanism by which meconium is expelled from the large bowel into the amniotic fluid is by release of the arginine vasopressin(AVP), from the fetal pituitary secondary to hypoxia. This AVP released causes the smooth muscle of colon to contract, resulting in intra amniotic defeacation.

Meconium spiration syndrome (MAS) a term used to describe respiratory distress in a meconium stained infant, is defined as the presence of meconium below the vocal cords. It was thought to occur with the infants' first breath.

MAS is defined by the following criteria:

1. Presence of meconium below the vocal cords
2. Clinical respiratory distress in the first 24 hours of life
3. Abnormal chest x-ray consistent with aspiration pneumonitis

Fetus exhibits breathing like movements in utero, but it is generally stated that amniotic fluid is normally swallowed and does not enter trachea, except during severe hypoxia so an episode of severe asphyxia is important in the production of MAS.

Mechanism:

When fetal distress occurs in the presence of meconium, the following events may result in meconium aspiration. Fetal asphyxia may cause pulmonary vasoconstriction and reduced pulmonary blood flow. Fetal gasping occurs

secondary to asphyxiated insult with aspiration of meconium into the trachea. With the reduction of pulmonary fluids, the self cleaning action of the trachea bronchial tree is lost and meconium remains in the trachea with potential for aspiration. In most of the cases aspiration of meconium into the peripheral airway is a postnatal event.

Release of the chest compression at birth may move meconium deeper and spontaneous breathing and mechanical ventilation would cause further peripheral dissemination. Incidence of MAS is 1-3% of those infants born with meconium stained liquor.

PATHOPHYSIOLOGY OF MAS

Meconium aspiration is likely to cause both mechanical obstruction of the airways and chemical pneumonitis. The free fatty acids in the meconium will strip away alveolar surfactant (Clark and Colleagues-1987). Atelectasis, consolidation, pneumothorax and pneumomediastinum may occur and prove rapidly fatal. Meconium has definite toxic properties of a low grade nature, similar to those of bile, but much more pronounced in their local effects. The pH of meconium ranges from both 5.5 – 7.0 Drisscoll and Smith felt that the irritating action of meconium on the pulmonary parenchyma might initiate a chemical pneumonitis, helping to compromise pulmonary function and this could explain the inflammatory changes

seen histologically in infants dying of MAS and partially explain the high incidence of effusion seen on chest roentgenograms.

Mechanical airway obstruction by particles of meconium or by squamous epithelial cells probably plays the most important role in the pathophysiology of MAS. A large amount of meconium is capable of completely obstructing the trachea, resulting in rapid death from asphyxia acute cor pulmonale. Smaller amounts move quickly to the lung periphery, resulting in obstruction of the distal airways. With complete obstruction of the peripheral airways, atelectasis of alveoli distant to the obstruction occurs.

Those collapsed alveoli that remain perfused cause right to left shunting of blood. Partial airway obstruction, on the other hand, would produce a ball valve effect. However, as the airway collapses around its obstruction during expiration this air remains trapped distally. If this trapped air builds up pressure or if intrathoracic pressure abruptly rises as during forced expiration, some air might leak out of the alveoli into the intestinal tissues of the lung. This causes the extra alveolar air, pneumothorax and pneumomediastinum.

Arterial desaturation:

It is the most important and consistent finding in meconium aspiration syndrome which occurs due to right to left shunts. These shunts occur at both cardiac level and the pulmonary level. Cardiac catheterization studies have shown

that shunts occur both through the foramen ovale and patent ductus arteriosus.

Persistent fetal circulation in MAS:

In severe MAS some infants develop right to left shunting of blood at the cardiac level returning to the fetal type of circulation.

Fox and co workers felt that the cause for persistence of fetal circulation with perinatal aspiration syndrome was due to chronic intra uterine asphyxia with secondary pulmonary arteriolar medial hyperplasia or hypertrophy. So recognition and treatment of pulmonary hypertension is important. The significance of passage of meconium is, however, questionable and is referred to as a murky subject (Katz & Bowes, 1992). Various clinical trials have been carried out in an effort to judge the significance of meconium passage in labour and to establish management guidelines but no definite consensus has been reached.

Abramovici et al (1974) compared perinatal outcome in 80 patients with meconium to 80 control parturients in whom amniotic fluid was clear. The mean Apgar score in the two groups was 8.58 and 8.57 respectively. The mean pH was 7.3 in those with thin meconium and 7.31 in those with thick meconium. There was no significant difference in terms of fetal pH, Apgar score and perinatal outcome in two groups. The author concluded that the passage of meconium could be a state of temporary fetal distress and delivery within a reasonable period of time ensures good outcome.

In an attempt to find out the significance of meconium during labour, continuous fetal heart rate monitoring and routine fetal scalp blood sampling was utilized in evaluation of 366 fetuses during labour out of which 106 had meconium at some stage of labour (Miller et al 1975). Although there was a significant fall in Apgar score at 5 minutes in thick meconium group (mean ± S.D.= 8.1 ± 1.5) versus no meconium group(mean ± S.D. = 8.8 ± 1.0) ($p < 0.05$), there was no statistically significant difference in signs of fetal distress attributable to the meconium per se.

The authors concluded that in the event of meconium passage, active intervention is indicated only when this is accompanied by > 10% late deceleration and fetal acidosis. The combination of fetal asphyxia and meconium staining of amniotic fluid enhances the potential for meconium aspiration and poor neo natal outcome.

A similar prospective study was undertaken to determine the passage of meconium during early labour (cervical dilatation ≤ 3 cm), the type of meconium (thin / thick) and fetal pH values could be correlated with Apgar score as predictor of neo natal outcome (Starks, 1980). Comparison was made between 177 patients with meconium staining and 100 patients without meconium staining. The thick meconium group had significant lower 1 and 5 minute Apgar scores and scalp blood pH as compared to thin meconium or no meconium group. (Table 7 & 8)

Table 7: Correlation between Apgar Score and presence of meconium stained liquor (Starks 1980)

Apgar score	**Thick Meconium n = 101**	**Thin Meconium n = 76**	**No Meconium n = 20**
At 1 minute			
0-3	15	02	01
4-6	50	04	07
7-10	36	70	92
At 5 minutes			
0-3	01	0	0
4-6	08	0	01
7-10	91	76	99

Table 8: Correlation between Scalp Blood pH and presence of meconium stained liquor (Starks 1980)

Scalp Blood pH	**Thick Meconium n = 101**	**Thin Meconium n = 76**	**No Meconium n = 20**
< 7.25	15	0	01
> 7.25	86	76	19

In those patients with thick meconium, the presence of fetal heart rate abnormality, increase the perinatal morbidity markedly. The authors concluded that the early passage of thick meconium does correlate with adverse

fetal outcome and perinatal morbidity.

Mitchell et al (1985) also stated that meconium in labour is associated with increased perinatal morbidity and 53% of pregnant women with moderate to thick meconium stained liquor in their study had arterial pH less than 7.25 at delivery. Most of this acidosis was of metabolic type indicating a fundamental chronic problem.

There was no significant difference in fetal heart rate tracing between acidotic and non-acidotic group indicating poor predictive power of fetal heart rate tracings in the presence of meconium. The authors concluded that the presence of thick meconium by itself is an important intra partum risk sign and warrants early intervention.

Hence although most authors feel that meconium in labour indicates some degree of fetal distress, no definite opinion regarding management protocol based on the presence of meconium alone has been reached as yet. The relationship between fetal asphyxia as expressed by umbilical arterial blood gas measurement with meconium in amniotic fluid and Apgar score has also been studied earlier.

Low (1988) conducted a study on 1773 patients. Fetal asphyxia (defined as an umbilical arterial buffer base less than 34 mmol / I. was present in 39 fetuses(2.2%) thick meconium staining of liquor was seen in 262 patients (15%) correlation of umbilical arterial buffer base with presence of meconium and Apgar

score was studied.(table 9)

Table 9: Correlation of umbilical arterial buffer base, meconium and Apgar score (Low 1988)

Buffer base in mmol/L	Meconium in liquor(n=262)	Apgar score of 0-3	
		At 1 minute n=115	At 5 minutes n=11
<34(n==39)	12	18	03
> 34 (n=1734)	250	97	08

Table 10: Sensitivity and false positive rates of meconium and Apgar score in estimating fetal asphyxia (Low, 1988)

Clinical parameter	Sensitivity of detecting fetal asphyxia	False positive rates
Presence of meconium	32%	95%
APGAR score 0-3		
At 1 minute	46%	84%
At 5 minute	8%	73%

The authors concluded that, although there is an association between meconium, low Apgar score and Fetal asphyxia, these are not sensitive markers of fetal asphyxia.

Gilstrap et al (1989) attempted to precisely define birth asphyxia based on fetal condition at birth as measured by umbilical artery blood pH, Apgar score and neurological condition of the new born. He concluded that infants must be

severely depressed at delivery for birth asphyxia to be diagnosed. Such depression included an Apgar score of <. 3 at 1 and 5 minute in addition to umbilical arterial pH value of < 7.0.

Patho physiology of perinatal asphyxia:

Fetal asphyxia primarily results from impaired placental exchange. 5 principal mechanisms of perinatal asphyxia have been described (Stone & Murray, 1996).

1. Interruption of umbilical circulation (Cord compression / accidents)
2. Inadequate perfusion of maternal side of placenta (maternal hypotension/ hypertension/ abnormal uterine contractions).
3. Altered placental gas exchange (abruptio placentae/placenta previa/ placental insufficiency).
4. Impaired maternal oxygenation (cardio pulmonary disease/anaemia).
5. Failure of neonate to accomplish lung inflation and transition from fetal to neo natal cardio pulmonary circulation.

The initial respiratory insufficiency translates into hypoxemia, which leads to Tissue hypoxia and metabolic acidosis. The early outcome major like Apgar score or late out come measures such as motor or cognitive deficits have an unpredictable association with fetal metabolic acidosis since they failed to recognize the complexity of compensatory mechanism of the fetal asphyxia. The

effect of hypoxic event on the fetus is influenced by the patron of development and duration of asphyxia and nature of the fetal response (Low, 1988).

The fetus responds to asphyxia in two main ways: one is centralization of fetal circulation with increase blood flow to the brain, heart and adrenal. The second mechanism is increased erythropoiesis with influx of immature red blood cells into circulation to act as oxygen carriers (Low, 1988). This forms the basis of increased number of nucleated red blood cells following hypoxia.

Nucleated Red Blood cells / 100 white blood cells in umbilical venous blood as a predictor of perinatal asphyxia. The presence of nucleated RBCs in cord blood of neonates was first noted in 1871(Neumann & Anderson, 1941). Until the 6th & 7th week of gestation practically all the fetal Red blood cells are nucleated by the 12th week, their levels start declining and are uncommon at term, making their significance at term a matter of controversy.

The normal values of nucleated RBCs per 100 WBCs in term non asphyxiated new born is quite variable although rarely > 10 (Table 11)

Table 11: Normal values of nucleated RBCs / 100 WBCs in umbilical venous blood in different studies

Authors	No. of patients	Mean nucleated RBCs/ 100 WBCs	S.D
Sinha et al (1972)	84	2.3	0.69
Shiv hare et al (1976)	33	4.1	2.4
Green & Mimouni (1990)	102	1.7	6.2
Phelan et al (1995)	83	3.4	3.0
Hanlon Lundberg et al (1997)	1112	8.55	10.27
Hanlon Lundberg & Kirby (1999)	1561	9.2	18.1

Before attributing high levels of nucleated RBCs to an acute intra partum asphyxial event, it is imperative to rule out other conditions which may cause a rise in the level independently.

Hanlon Lundberg et al (1997) showed that the infants of diabetic mothers had polycythemia and resultant significant higher level of nucleated RBCs/ 100 WBCs in cord blood even in the absence of any acidemia [14.62 12.24 versus 8.32 ± 10.13 ($p<0.01$)].

Green and Mimouni (1990) in a similar study reported higher levels of nucleated RBCs / 100 WBCs in infants of diabetic mothers. The term neonates with no high risk factors had 1.7 ± 6.2 nucleated RBCs/100 WBCs whereas in

those born to diabetic mothers the corresponding levels were 13.0 ± 18.9 & 8.3 ± 17.8 depending on presence or absence of any birth asphyxia. This was a significant difference with p value < 0.05.

That the levels of nucleated RBCs/100 WBCs are higher in pre term as compared to term neonates was shown by Philip & Tito, 1989 with significant higher level being observed in those fetuses who were small for gestation age as compared to appropriate for gestation age neonates. In the appropriate for gestation age group, those who delivered at 24 weeks had a higher level (mean ± S.D. = 71 ± 60) compared to those delivered at 33 weeks (mean ± S.D. = 26 ± 33). The percentage of small for gestation age newborns with either more than 40 or 100 nucleated RBCs/100 WBCs were 62% and 36% respectively. The corresponding percentages in the appropriate for gestation age were 25% & 6%. Both the differences were statistically significant ($p<0.05$)

Maier et al 1994, investigated the relationship between chronic fetal asphyxia and Erythropoietin levels. The levels were significantly higher in small for gestation age infants (median = 71.2 vs. 31.9 mill/ ml, $p<0.001$). Implicating higher Erythropoietin levels being associated with chronic placental insufficiency, Umbilical arterial pH being same in both the groups. The Erythropoietin levels at term correlated with level of nucleated RBCs ($r = 0.74$; $p < 0.002$) but not with hematocrit ($r = 0.18$); $p= 0.06$). There was also no significant correlation between

venous, hematocrit and nucleated RBCs, ($r = 0.14$; $p = 0.15$) the significant correlation between Erythropoietin level & nucleated RBCs count indicates that stimulation of fetal Erythropoietin occurs by the presence of chronic hypoxia (placental insufficiency).

In another similar study to show the effect of hypoxia on the fetus Snijders et al 1993 measured plasma erythropoietin level in 33 small for gestation age fetus between 26-38 weeks period of gestation. They showed significantly higher level than normal, when correlated for gestation age [Plasma Erythropoietin mean difference = 2.31 SD ($p<0.0001$)]. The umbilical arterial pH and p02 were also significantly reduced. There was an associated increase in Erythroblast count (mean difference = 3.56SD, $p>0.0001$) but the mean Erythrocyte count was in normal range. The author concluded that by measuring Erythroblasts an assessment of tissue oxygenation can be made.

Hanlon - Lundberg et al (1997) aimed to establish normal values of nucleated RBCs in term singletons and factors associated with their elevation. Cord blood was prospectively collected from 1112 patients. The mean + SD of nucleated RBCs / 100 WBCs was 8.55 ± 10.7 (Range 0-89). No significant difference was seen by maternal tobacco intake, drug abuse, Anemia, Fetal presentation and mode of delivery. Infants of diabetic mothers had significantly higher values (14.62 ± 12.24 vs. 8.32 ± 10.13, $p<0.01$). The levels of nucleated

RBCs/100 WBCs tended to be inversely proportional to the Apgar score. One minute Apgar scores of 0 -3 were significantly associated with higher counts than were Apgar scores of 7-10 (14.51 ± 14.65 Vs. 8.16 ± 9.78, $p< 0.05$) The same inverse proportionality was seen when nucleated RBCs were compared to pH (Table 12).

Table 12: Correlation of Arterial pH value and mean nucleated RBCs/100WBCs (Hanlon-Lundberg et a I, 1997)

pH	Mean nucleated RBCs /100 WBCs	SD	P value
7.10 to 7.19	12.4	12.15	<0.05
7.20 to 7.29	9.39	10.61	<0.01
7.30 to 7.39	6.8	8.29	-

The authors concluded that elevated nucleated RBC levels in Cord blood are associated with markers of intra uterine hypoxia such as meconium, lower Apgar scores and Acidemia. It can thus be used as a potential tool in estimating the degree and timing of intra uterine hypoxia. The added advantage of cord blood nucleated RBC estimation was that it could be obtained non-invasively from otherwise discarded specimen and analysed by personnel on equipment readily available in most hospital laboratories.

In a separate study Hanlon-Lundberg and Kirby, (1999) evaluated 1561 cases to estimate the normal nucleated RBC levels in term newborns. The mean nucleated RBCs/100WBCs was 9.2 + 18.1 with a wide range of 0-327. The levels were significantly higher than pH was <7.20 ($p = 0.001$), meconium was present ($p = 0.020$) and when there was neonatal intensive care unit admission, ($p = 0.024$).

Phelan et al (1995) conducted a study to determine whether a relationship exists between the presence of nucelated RBCs, development of hypoxic ischaemic encephalopathy and long term neurological development. They compared the nuceated RBC levels in cord blood from 46 term neurologically impaired neonates to 83 non asphyxiated newborns. The first and highest nucleated RBC levels and the time for disappearance was assessed. The neurologically impaired group had significant higher levels (34.5 ± 68.5) as compared to the control group. (3.4 ± 3.0, $p<0.0000$). It was also seen that greater the time interval between the hypoxic events and birth, higher was the number of nucleatic RBCs which also had delayed clearance from the circulation.

Thilaganathan et al (1994) established a relationship between umbilical arterial pH, Apgar Score, Leucocyte count and Erythroblasts count at birth in three groups of singleton pregnancies delivered at term vaginally (n = 55) by elective caesarean section (n = 39) or by emergency caesarean section (n = 55) (Table 16)

Table 13: Correlation between mode of delivery, pH value, Erythroblasts and Leucocyte count [Thilaganathan et al (1994)]

Parameter	**Elective caesarean section (n = 39)**	**S. Vaginal delivery (n=55)**	**Emergency caesarean section (n=55)**
Umbilical Arterial pH: median (range)	7.26(7.08-7.37)	7.26 (7.0-7.38)	7.22 (6.91-7.4)
Erythroblast count (x 10 10^9/L)	0.30(0.00-0.49)	0.75 (0.0-5.3)	1.1 (0.00-15.9)
Leucocyte count (x 10^9/L)	10.6(6.2-17.7)	13.8(7.25-48.0)	13.5(4.2-40.3)

In the emergency caesarean section group the umbilical arterial pH was significantly lower and the leucocytes and Erythroblasts counts were higher than in the elective caesarean section groups. Comparison of emergency caesarean section & spontaneous vaginal delivery groups showed significant differences in cord blood pH and Erythroblasts count but not for the leucocytes counts. IN the spontaneous vaginal delivery group itself erythroblastosis was associated with umbilical arterial pH ($R=0.543$; $p<0.0002$) whereas leucocytosis was associated with length of labour ($R = 0.309$; $p<0.05$). The five minute Apgar score was more than or equal to 7 in all infants. This study suggested that Leucocytosis is a non

specific response of the fetus to labour, whereas erythroblastosis reflects fetal tissue hypoxia.

Baschat et al (1999) sought to determine a relationship between the nucleated RBC level alongwith other hematological parameters and IUGR with hypoxemia in 84 fetuses. All the fetuses evaluated had an elevated umbilical artery pulsatility index (more than 2 SD above means) and a subsequent birth weight <10th percentile. The fetuses were grouped as:

1. Elevated umbilical artery pulsatility index only.

2. Middle cerebral artery pulsatility index > 2SD below the gestational age mean in addition to elevated umbilical artery pulsatility index.

3. Either peak velocity index > 2SD above the gestational age mean in the inferior venacava and ductus venosus or pulsatile flow in the umbilical vein or both.

A neonatal full blood count including nucleated RBC was performed with one hour of delivery followed by serial daily measures till the level of nucleated RBC count was < 5/100 WBC. (Table 14)

Table 14: (Correlation between nucleated RBC & other hematological parameters (Baschat et al, 1999)

Hematological parameter	**Group I (n = 32)**	**Group II (n = 16)**	**Group III (n = 36)**
Nucleated RBC/100WBC median (range)	8.5 (1-220)	38.5 (1-273)	145 (2-3180)
Platelet count (x $l0^3$)	204.5 (63-412)	168 (101-410)	142 (42-451)
Haemoglobin (gm/dl)	17.1 (14.7-22.1)	15.9 (13-21.6)	15.3 (8.1 -20.1)
Hematocrit (%)	54.1 (44.1-68.4)	50.2 (40.1-71.5)	49.4 28.2-74.0)
WBC count (x $l0^3$)	9.6 (2.7 - 14.4)	8.3 (2.5-14.0)	4.5 (1.7 - 19.7)

The nucleated RBC counts were higher in Groups II and III than in Group I.

$p<0.05$ and <0.005 respectively (the persistence of nucleated RBC in circulation was also longer in Group II & III. Neonates in Group III also had lower platelet count, Hb value, Hematocrit level and WBC count. This finding of lower Hb and Hct values seem to contradict the concept of secondary polycythemia in response to chronic hypoxia. The author explained these findings by the hypothesis that intra placental thrombosis in chronic placental insufficiency leads to platelet and erythrocyte consumption. The author also found that umbilical artery bicarbonate HCo3 level rather than PO2 was the strongest determinant of the peak nucleated

RBC level and persistence of nucleated RBC elevation. $X2=0.27$, $p< 0.001$ and $X2 = 0.47$, $p<0.0001$.

HAEMATOLOGICAL INDICATOR OF FETAL ACIDEMIA

Nucleated red blood cells have been implicated as a possible marker of perinatal brain damage. Ever since the presence of nucleated red cells in the peripheric blood of the newborn infant, became known, their significance has been a matter of dispute.

The first investigations as to the presence of nucleated red cells in the blood of the new born infant are accredited to Neumann in 1871. According to Lipprnan, he was not impressed by the number of erythroblasts in the normal full term infant, but he had noticed their presence in premature infants in 1895. Gerssler & Japha concluded that normoblast did not occur in the blood of the normal newborn infant and their presence therefore must be considered as a pathologic sign.

Another study showed normoblasts in the blood of the newborn full term infant are not a physiologic phenomenon in the strict sense, but rather conditioned by an abnormal act of delivery. They also noted immature white cells parallel to an increase of nucleated red cells in the first day of life. Conversely, in the absence of immature white cells, nucleated red cells were not present or only in very small numbers. These authors interpreted these cells as signs of, or reactive phenomena

to, tissue damage, especially during the act of delivery. Disintegration of tissue and blood which is avidly resorbed in this early period of life, leads to a noticeable increase of white cells and especially to the liberation of nucleated red blood cells.

These abnormal cells disappeared from circulation with in 24 to 48 hours. Their elaborate study lead them to postulate a “birth crisis” in the blood picture of the newborn infant, which they feel, is conditioned by tissue damages during the course of delivery.

According to Javert, a certain number of NRBCs (10 or more) per 100 white blood cells is in itself a sign of prematurity, regardless of whether or not weight, length and duration of pregnancy are also pointing to prematurity. That high nucleated red cells counts might be caused by an asphyxic condition, as for example in congenital heart disease. They noted that in the blood smears from cases with different abnormalities and from asphyxiated babies in general, an increased number of nucleated erythrocytes was found.

Rolf et al investigated the relationship between erythropoetin concentration in umbilical venous blood and clinical signs of asphyxia; and reported that elevated erythropoietin concentrations in umbilical venous blood indicate prolonged fetal hypoxia.

Another study of elevated NRBC count in neonatal blood and Doppler detected circulatory decompensation in fetuses with intrauterine growth restriction

are associated with hypoxemia. They sought to determine the relationship between nucleated red blood cell count at birth and the circulatory status of fetuses with intrauterine growth restriction. Increasing abnormality and venous flow in fetuses with intrauterine growth restriction is associated with increased NRBC count at birth. Metabolic acidemia associated with asphyxiated state appears to be the main determinant of the rise in nucleated red blood cells.

Kathleen M Hanlon Lundberg et al aimed to establish normal values for nucleated red blood cells in term singletons and factors associated with their elevation. They collected cord blood from singleton gestations prospectively. Umbilical vein WBCs and NRBCs were counted and umbilical arterial pH was determined. The mean number of nucleated red blood cells per 100 WBCs was 8.55, with a wide range and SD. Elevated values were associated with markers of intrauterine hypoxia such as meconium, lower apgar scores and lower pH values.

Kathleen studied the relationship between nucleated red blood cell count in the circulation of term neonates and other possible markers of acidemia and found that NRBCs counts vary widely in the circulation of term neonates. Elevated NRBC count are associated with fetal acidemia, meconium and neonatal intensive care unit admission.

Acute asphyxia

Acute asphyxia is the traditional cause of failure to breathe at delivery, and is probably the mechanism responsible for babies not breathing at birth after they have suffered some acute crisis, such as an antepartum haemorrhage or cord prolapse. The neonate is capable of surviving at least 20 minutes of complete oxygen deprivation, but in the latter part of this period brain damage can occur.

Elevated nucleated red blood cell counts after intrauterine hypoxemia have been noted in the absence of reticulocytosis. Because the reticulocyte response to erythropoietin may require 2-3 days. It has been suggested that different hypoxia - associated mechanisms may account for the rise in nucleated red blood cell count.

Epinephrine, an acute stress hormone, may modulate erythropoiesis and could therefore stimulate nucleated red blood cell release. Acute fetal hypoxemia causes nucleated red blood cell release and lymphocytosis in as few as 2 hours certainly unrelated to erythropoiesis.

MATERIALS AND METHODS

This study was done in Govt kilpauk medical college, Chennai .Around 320 patients who have undergone emergency LSCS irrespective of indication have been taken to know NRBC's (nucleated redblood cells /100WBC's) as a indicator of perinatal asphyxia.

Ethical committee approval obtained and enclosed

STUDY DESIGN: Prospective Cross sectional study

PERIOD OF STUDY: April 2014 to September 2014

STUDY GROUP:

Singleton term pregnancies primi /multi babies of more than 2.5kg appropriate for gestational age delivered by emergency lscs irrespective of indication without any maternal co morbid factors.

INCLUSION CRITERIA:

- Singleton Term Pregnancies
- Primi /Multi
- Babies of more than 2.5kg
- Appropriate for gestational age
- Emergency LSCS

EXCLUSION CRITERIA:

Pregnancies known to be associated with

- Women with Rh isoimmunization
- Women with gestational diabetes mellitus
- Post term pregnancy
- IUGR
- Pre eclampsia patients
- Newborn with congenital anomalies
- Preterm babies

SAMPLE SIZE: 320

Sample size was determined on the basis of a pilot study in which the incidence of Birth Asphyxia was measured as 28%. We calculated a minimum sample size of 310, assuming a type 1 error (two-tailed) of 0.05 and a margin of error of 5%.

$$\mathbf{n} = \frac{\mathbf{t^2} \times \mathbf{p(1-p)}}{\mathbf{m^2}}$$

Description: **n** = required sample size

t = confidence level at 95% (standard value of 1.96)

p = estimated prevalence of malnutrition in the project area

m = margin of error at 5% (standard value of 0.05)

$$n = \frac{(1.96)^2 \times 0.28(1-0.72)}{(0.05)^2}$$

$$n = \frac{3.8146 \times 0.2016}{0.0025}$$

$$= 309.78$$

$$= 310$$

METHODOLOGY:

• Sample was processed immediately. In case of any delay between the time of collection of sample and the timing of taking the reading, the samples were refrigerated.

PROCEDURE:

- From all subjects, samples of cord blood collected immediately after clamping and cutting the umbilical cord.
- Patients Name, Age, IP NO, Time of collection have been written on the test tube
- Sample taken in EDTA coated bottle for purpose of making smears.
- 2 ml of blood was collected

- For making smear, two clear glass slides were taken and a drop of sample was placed towards one end.
- A spreader glass slide placed at 45^0 inclination to sample and in one uniform motion drop of blood smeared on rest of slide.
- Slide is allowed to dry and then covered with Leishman's stain. After 5 minutes stain is diluted with distilled water and mixed on slide.
- Slide is allowed to take in stain for 15 minutes and then washed in gentle stream of water.
- Under pathologist's supervision, smear focused under high power microscope and RBCs (nucleated) counted against 100 WBCs.
- A thin smear was made of the umbilical venous blood and stained with Leishmans's stain The smear was studied under 45x magnification and number of nucleated red blood cells/100 white blood cells was determined by scanning the film from one end till 100 WBC's were counted.
- The nucleated RBC count of cord blood was determined.

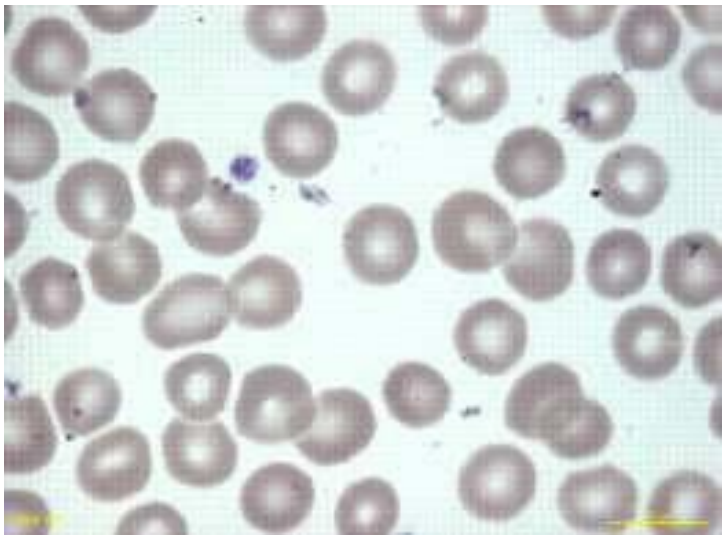


Figure 10: Peripheral smear showing normal RBC's

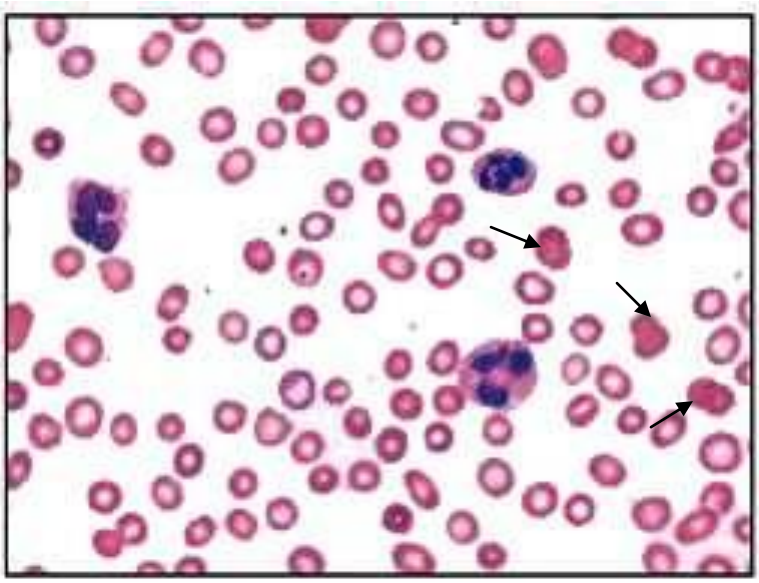


Figure11: Peripheral smear showing nucleated RBC's

Follow up

The number of neonates admitted to NICU or who had developed evidence of HIE was noted according to modified sarnat staging, correlating the number of NRBC's with neonatal outcome.

TABLE 15: MODIFIED SARNAT STAGING FOR HIE

STAGE	**STAGE 1**	**STAGE 2**	**STAGE 3**
Level of consciousness	Hyperalert	Lethargic/obtunded	Stupor or coma
Activity	Normal	Decreased	Absent
Neuro muscular control: Mus tone	Normal	Mild hypotonia	Flaccid
Reflexes : Suckling	Weak	Weak/ absent	Absent
Autonomic function: Heart rate	Tachycardia	Bradycardia	Variable
Seizures	None	Common: focal/ multifocal	uncommon

RESULTS

Statistics

Descriptive statistics was done for all data and suitable statistical tests of comparison were done. Continuous variables were analysed with the unpaired t-test and categorical variables were analysed with the Chi-Square Test and Fisher Exact Test. Statistical significance was taken as $P < 0.05$. The data was analysed using EpiInfo software (7.1.0.6 version; Center for disease control, USA) and Microsoft Excel 2010.

The final sample selected was n= 274 in group A and n= 46 in group B

Treatment Groups

TABLE 16: Table showing outcome of study groups

Treatment Groups	Name of the Group	Treatment	Number of Subjects
Group A	No Asphyxia	Singleton term pregnancies primi /multi babies of more than 2.5kg appropriate for gestational age delivered by emergency lscs irrespective of indication without any maternal co morbid factors without Birth Asphyxia	274
Group B	Asphyxia +	Singleton term pregnancies primi /multi babies of more than 2.5kg appropriate for gestational age delivered by emergency lscs irrespective of indication without any maternal co morbid factors with Birth Asphyxia	46

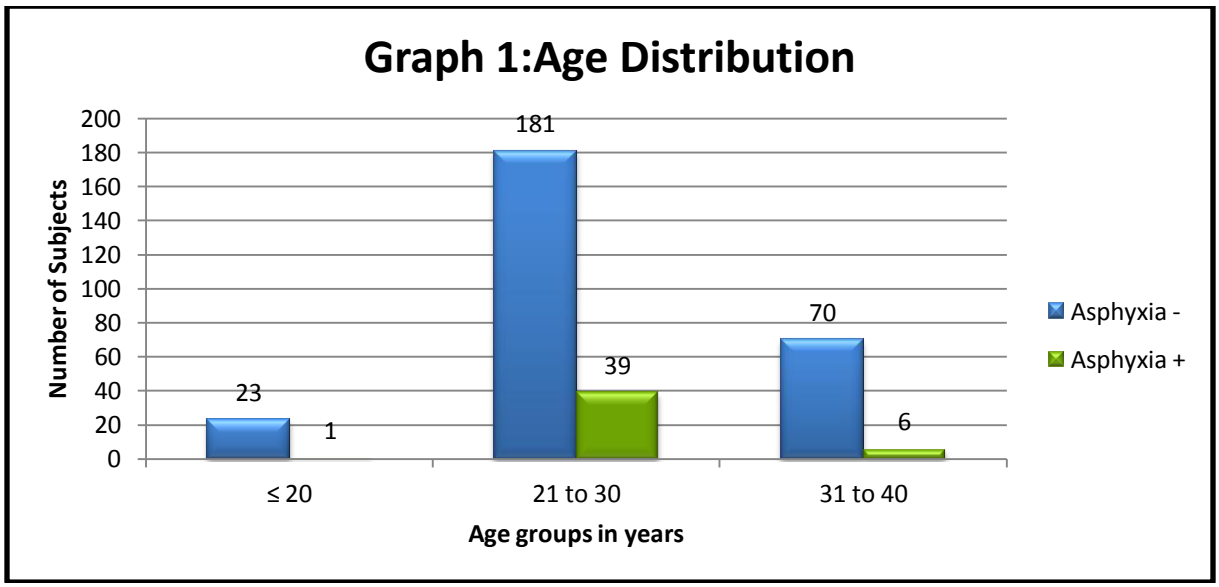
In this study out of 320 babies 46 babies have diagnosed by the paediatrician as birth asphyxia got admitted in NICU and remaining 274 babies were diagnosed as no asphyxia transfer to mother side.

Age

TABLE 17: Age group wise distribution of study population

Age Distribution	No Asphyxia	%	Asphyxia +	%
≤ 20	23	8.39	1	2.17
21 to 30	181	66.06	39	84.78
31 to 40	70	25.55	6	13.04
Total	274	100	46	100

Age Distribution	No Asphyxia	Asphyxia +
N	274	46
Mean	27.83942	26.43478
SD	4.573748	5.044919
P value Unpaired t – test	0.081943	



By conventional criteria the association between the birth asphyxia and age is considered to be not statistically significant since $p > 0.05$. Since age is not statistically significant, it means that there is no difference between the groups. In other words the groups contain subjects with the same basic demographic characteristics.

Gravida

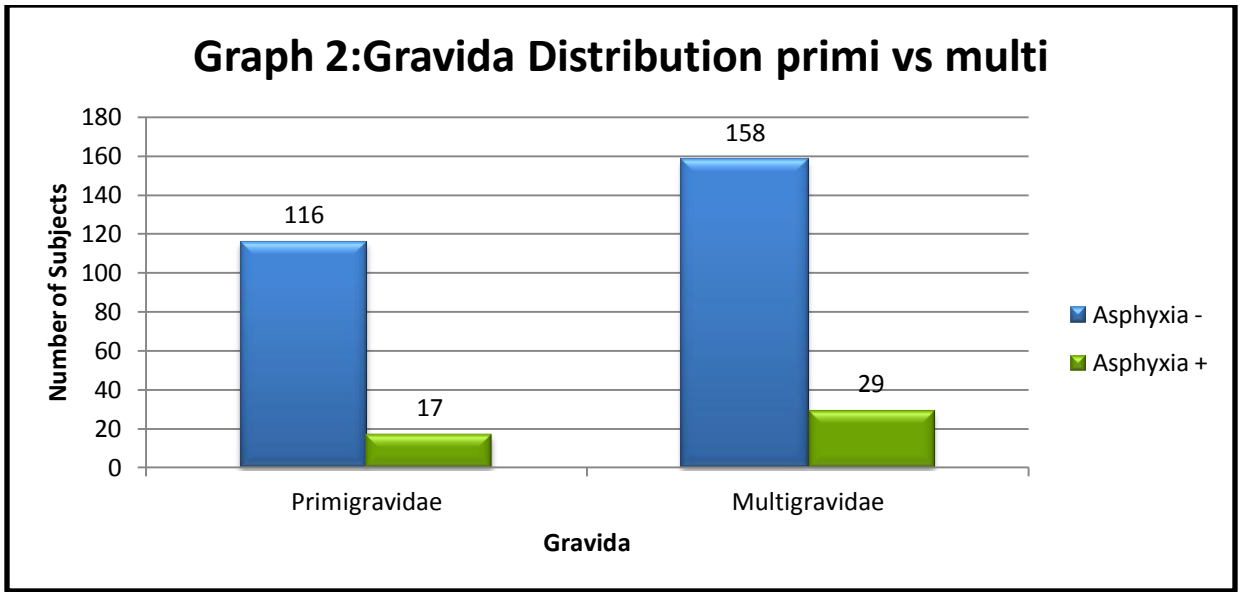
Table 18: Percentage distribution of study population according to gravida

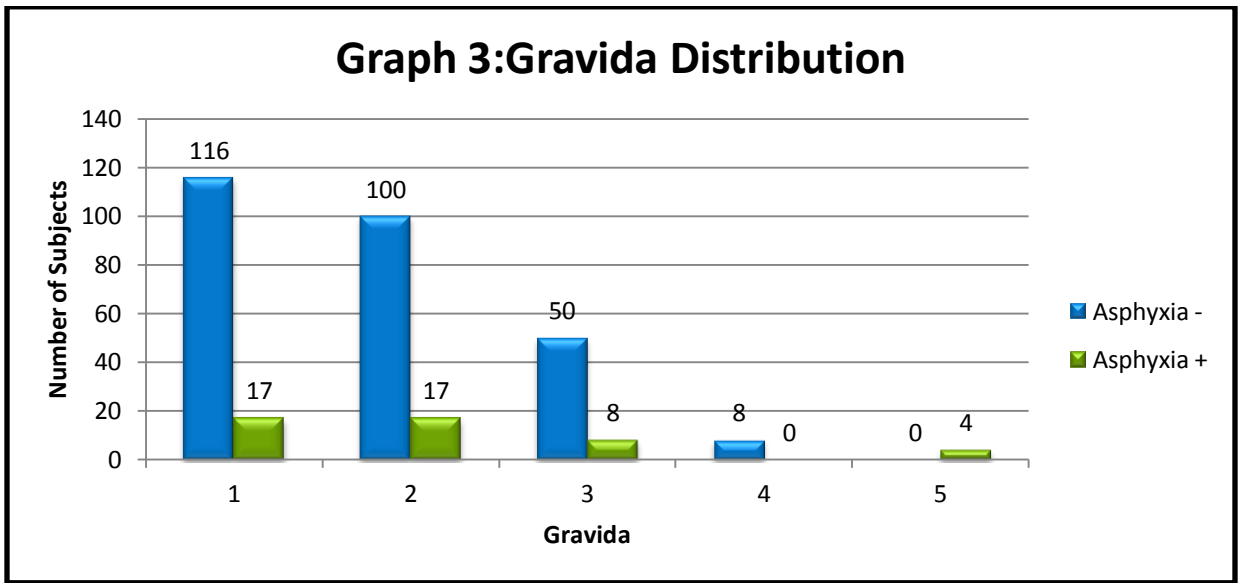
Gravida	No Asphyxia	%	Asphyxia +	%
1	116	42.34	17	36.96
2	100	36.50	17	36.96
3	50	18.25	8	17.39
4	8	2.92	0	0.00
5	0	0.00	4	8.70
Total	274	100	46	100

Gravida	No Asphyxia	%	Asphyxia +	%
Primigravidae	116	42.34	17	36.96
Multigravidae	158	57.66	29	63.04
Total	274	100	46	100

	Primigravidae	%	Multigravidae	%
No Asphyxia	116	87.22	158	84.49
Asphyxia +	17	12.78	29	15.51
Total	133	100	187	100

Chi-squared Test	
chi-squared statistic	0.469
Degrees of Freedom	1
P value	0.4933





By conventional criteria the association between the birth asphyxia and Gravida is considered to be not statistically significant since $p > 0.05$.

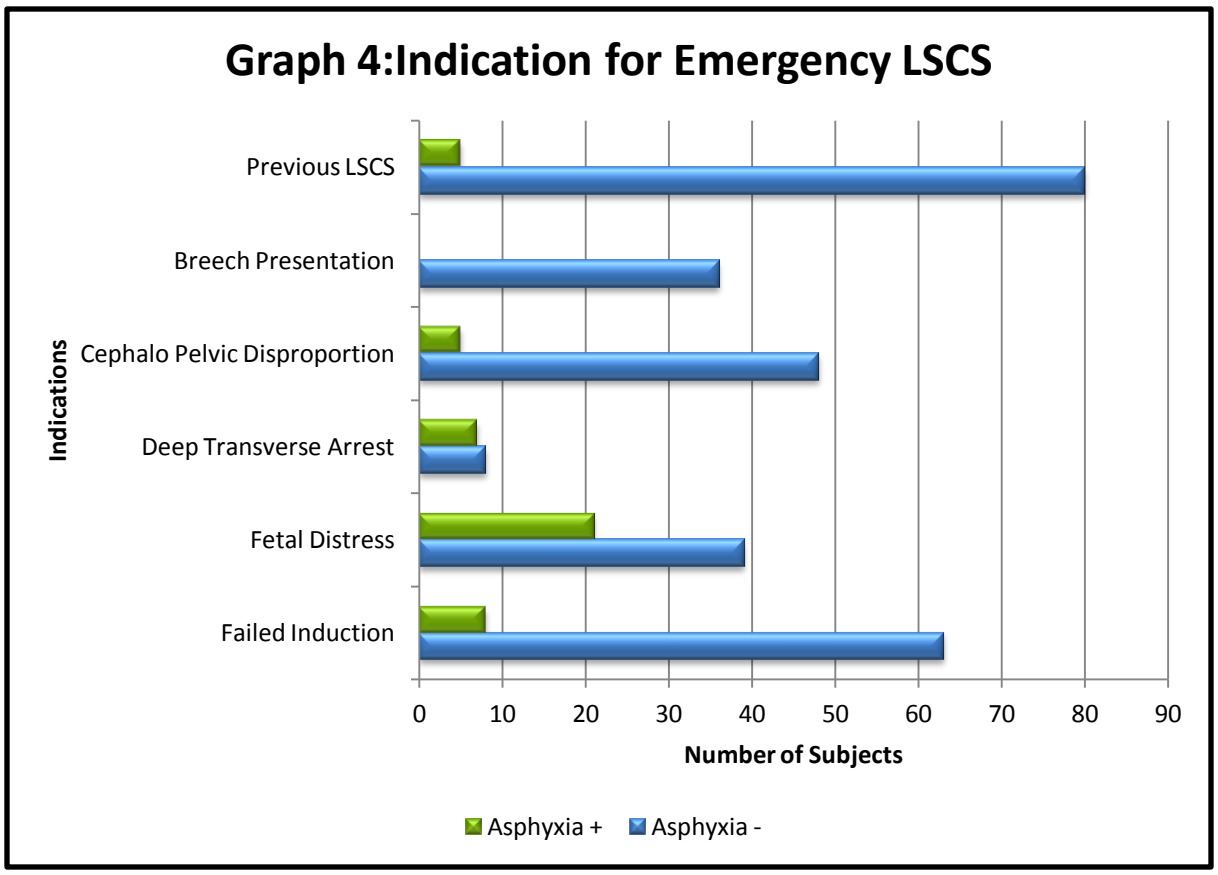
Indication for Emergency LSCS

Table 19: Distribution of study population according to indication of emergency LSCS

Indication for Emergency LSCS	No asphyxia	%	Asphyxia +	%
Failed Induction	63	22.99	8	17.39
Fetal Distress	39	14.23	21	45.65
Deep Transverse Arrest	8	2.92	7	15.22
Cephalo Pelvic Disproportion	48	17.52	5	10.87
Breech Presentation	36	13.14	0	0.00
Previous LSCS	80	29.20	5	10.87
Total	274	100	46	100

Chi-squared Test	
chi-squared statistic	92.00
Degrees of Freedom	5
P value	0.000*

	Failed Induction	%	Fetal Distress	%	Deep Transverse	%	Cephalo Pelvic Disproportion	%	Breech Presentation	%	Previous LSCS	%
No asphyxia	63	88.73	39	65.00	8	53.33	48	90.57	36.00	100.00	80	94.12
Asphyxia +	8	11.27	21	35.00	7	46.67	5	9.43	0.00	0.00	5	5.88
Total	71	100	60	100	15	100	53	100	36	100	85	100



By conventional criteria the association between the birth asphyxia and Indications for Emergency LSCS (fetal distress and deep transverse arrest) is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. .

In simple terms, in emergency LSCS, the incidence of birth asphyxia is more when there is a fetal distress as an indication for emergency LSCS. It is statistically significant with a p-value of 0.00031 according to Chi-squared Test.

In simple terms, in emergency LSCS, the incidence of birth asphyxia is more when there is a deep transverse arrest as an indication for emergency LSCS. It is statistically significant with a p-value of 0.0367 according to Chi-squared Test.

Clinical Significance

The occurrence of birth asphyxia was meaningfully more (45.65%) when the indication for emergency LSCS was fetal distress, with 35% of the children with foetal distress finally suffering from asphyxia.

The occurrence of birth asphyxia was meaningfully more (15.22%) when the indication for emergency LSCS was deep transverse arrest, with 46.67% of the children born due to the complication of deep transverse arrest finally suffering from asphyxia.

This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is real intrapartum obstetric risk factor for developing Birth asphyxia if the indication for emergency LSCS was Foetal Distress and deep transverse arrest in our study. .

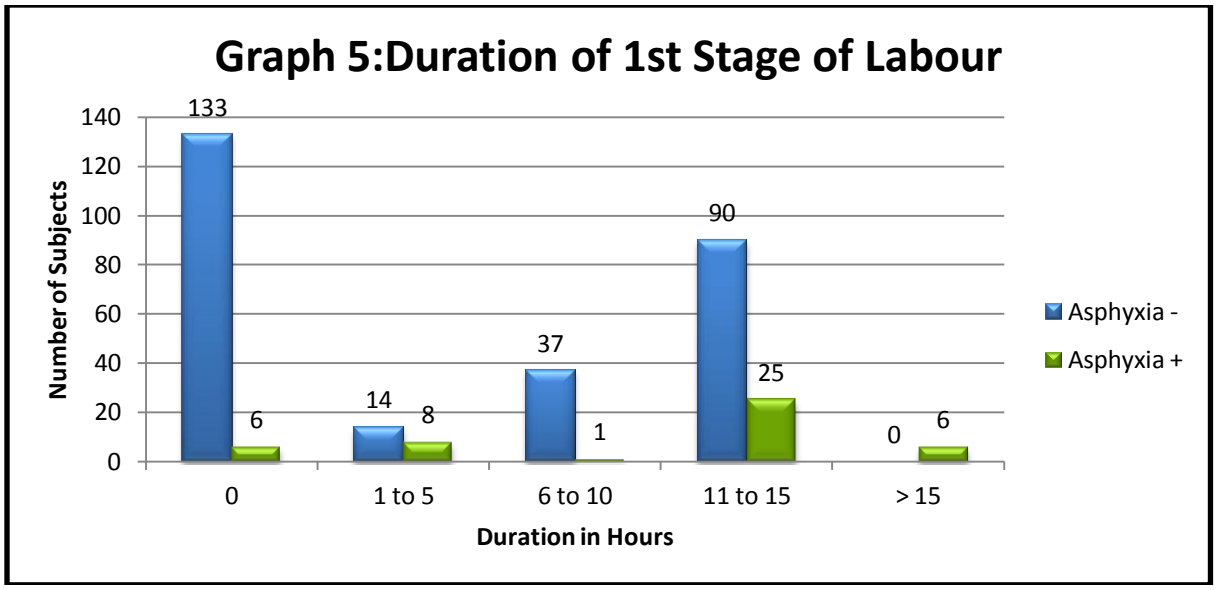
Duration of 1st Stage of Labour

Table 20: Distribution of study population according to duration of 1st stage of labour

Duration of 1st Stage of Labour in hours	No Asphyxia	%	Asphyxia +	%
0	133	48.54	6	13.04
1 to 5	14	5.11	8	17.39
6 to 10	37	13.50	1	2.17
11 to 15	90	32.85	25	54.35
> 15	0	0.00	6	13.04
Total	274	100	46	100

Duration of 1st Stage of Labour in hours	0 hr	%	1 to 5 hr	%	6 to 10 hr	%	11 to15 hr	%	> 15 hr	%
No Asphyxia	133	95.68	14	63.64	37	97.37	90	78.26	0	0.00
Asphyxia +	6	4.32	8	36.36	1	2.63	25	21.74	6	100.00
Total	139	100	22	100	38	100	115	100	6	100

Duration of 1st Stage of Labour	No Asphyxia	Asphyxia +
N	274	46
Mean	5.423358	9.826087
SD	2.862556	5.425472
P value Unpaired t - test	0.000*	



By conventional criteria the association between the birth asphyxia and duration of labour in 1st stage is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among the groups and the difference is significant. In simple terms, in emergency LSCS, the incidence of birth asphyxia is more when the duration of 1st stage of labour increased. It is statistically significant with a p-value of 0.000 according to unpaired t-test.

Clinical Significance

The average duration of 1st stage of labour was 9.83±5.43 hours in Asphyxia+ group compared to 5.42±2.86 hours in No Asphyxia group. The occurrence of birth asphyxia was meaningfully more (67.39%).when the the duration of 1st stage of

labour increased more than 10 hours. The duration of 1st stage of labour was meaningfully more (81%) in the Asphyxia+ group compared to No Asphyxia group by 4 hours and 24 minutes. This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is real intrapartum obstetric risk factor for developing birth asphyxia if the duration of 1st stage of labour exceeds 10 hours.

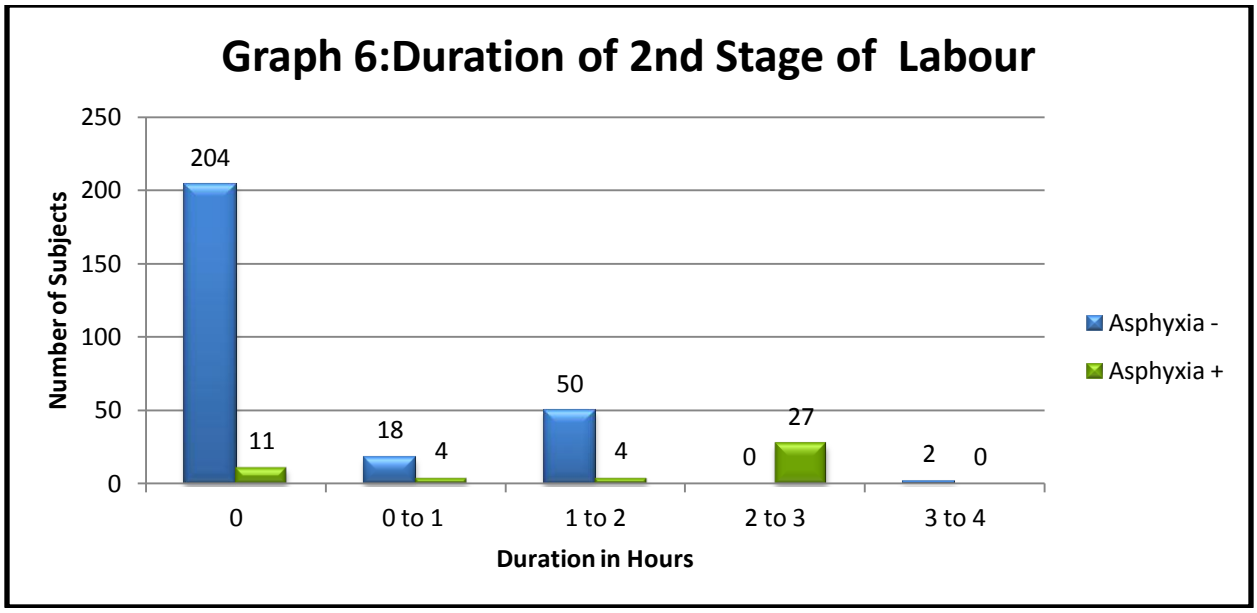
Duration of 2nd Stage of Labour

Table 21 : distribution of study population according to duration of 2nd stage of labour

Duration of 2nd Stage of Labour in hours	No Asphyxia	%	Asphyxia +	%
0	204	74.45	11	23.91
0 to 1	18	6.57	4	8.70
1 to 2	50	18.25	4	8.70
2 to 3	0	0.00	27	58.70
3 to 4	2	0.73	0	0.00
Total	274	100	46	100

Duration of 2nd Stage of Labour in hours	0 hr	%	0 to 1 hrs	%	1 to 2 hrs	%	2 to 3 hrs	%	3 to 4 hrs	%
No Asphyxia	204	94.88	18	81.82	50	92.59	0	0.00	2	100.00
Asphyxia +	11	5.12	4	18.18	4	7.41	27	100.00	0	0.00
Total	215	100	22	100	54	100	27	100	2	100

Duration of 2nd Stage of Labour	No Asphyxia	Asphyxia +
N	274	46
Mean	1.459854	2.352174
SD	0.838755	0.848983
P value Unpaired t - test	0.01595*	



By conventional criteria the association between the birth asphyxia and duration of labour in 2nd stage is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among the groups and the difference is significant. In simple terms, in emergency LSCS, the incidence of birth asphyxia is more when the duration of 2nd stage of labour increased. It is statistically significant with a p-value of 0.01595 according to unpaired t-test.

Clinical Significance

The average duration of 2nd stage of labour was 2.35±0.85 hours in Asphyxia+ group compared to 1.45±0.84 hours in No Asphyxia group. The occurrence of birth asphyxia was meaningfully more (58.70%).when the the duration of 2nd stage of

labour increased more than 2 hours. The duration of 2nd stage of labour was meaningfully more (62%) in the Asphyxia+ group compared to No Asphyxia- group by 54 minutes. This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is real intrapartum obstetric risk factor for developing Birth asphyxia if the duration of 2nd stage of labour exceeds 2 hours.

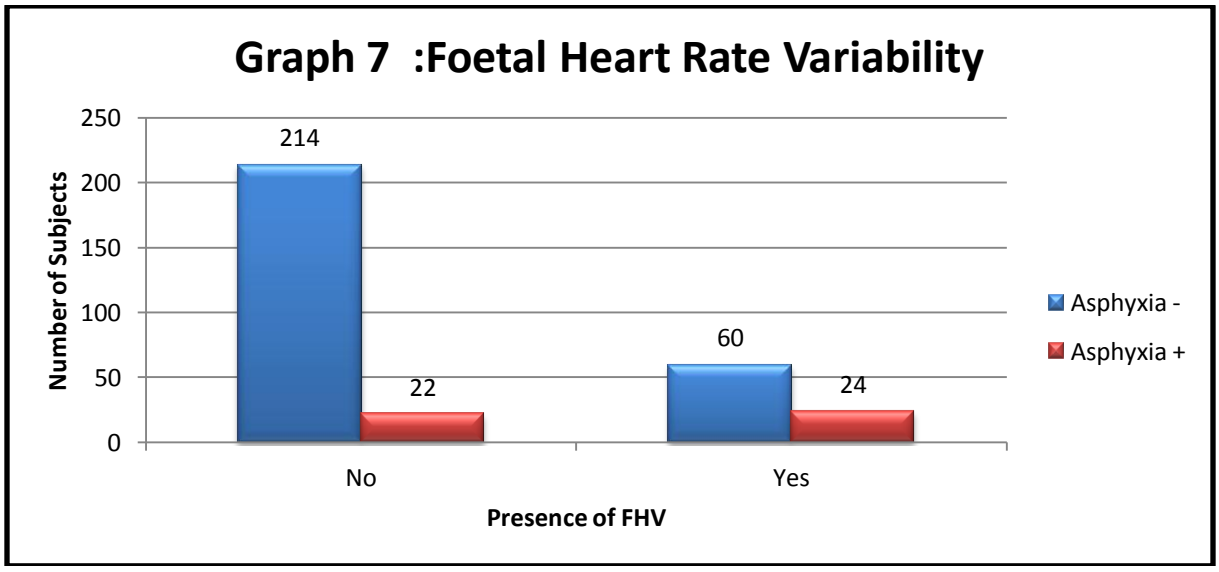
Fetal Heart Rate Variability

Table 22: Fetal heart rate variability of study participants

Fetal Heart Rate Variability	No Asphyxia	%	Asphyxia +	%
No	214	78.10	22	47.83
Yes	60	21.90	24	52.17
Total	274	100	46	100

Fetal Heart Rate Variabiliy	No	%	Yes	%
No Asphyxia	214	90.68	60	71.43
Asphyxia +	22	9.32	24	28.57
Total	236	100	84	100

Chi-squared Test	
chi-squared statistic	18.60
Degrees of Freedom	1
P value	0.000*



By conventional criteria the association between the birth asphyxia and fetal heart rate variability LSCS is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among groups and the difference is significant. . In simple terms, in emergency LSCS, the incidence of birth asphyxia is more when there is a fetal heart variability compared to absence of it. It is statistically significant with a p-value of 0.000 according to Chi-squared Test.

Clinical Significance

The occurrence of birth asphyxia was meaningfully more (52.17%) when there is presence of fetal heart rate variability compared to its absence (47.83%).

This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is real intrapartum obstetric risk factor for developing birth asphyxia if there is presence of fetal heart rate variability in our study. It can be used to predict early birth asphyxia.

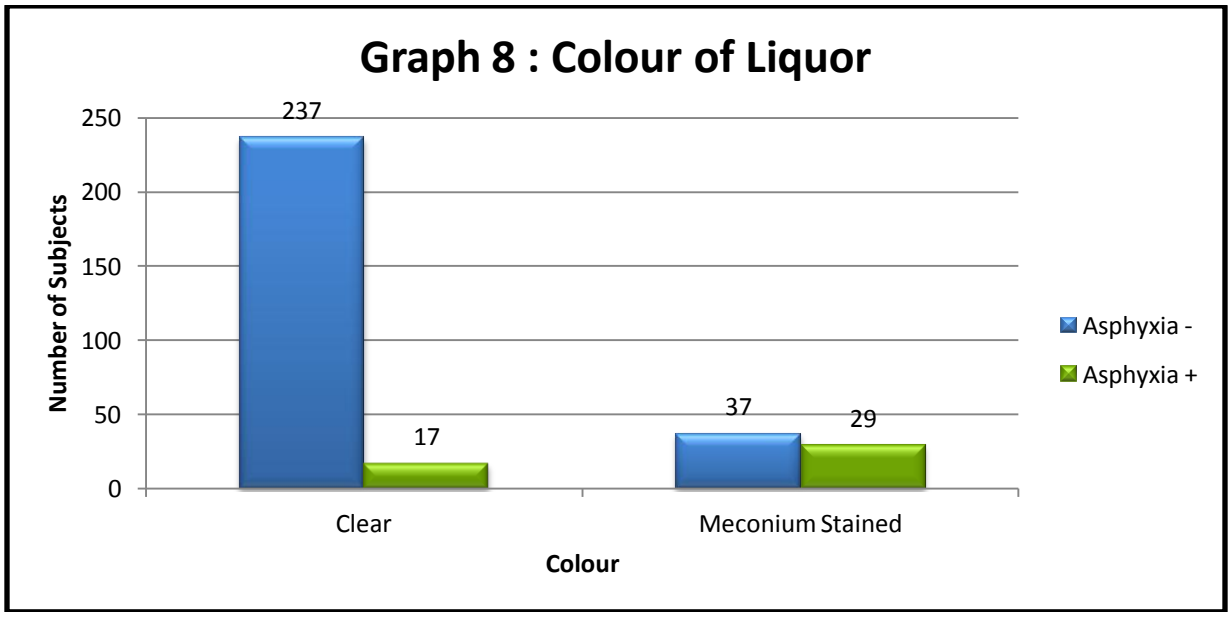
Colour of Liquor

Table 23 : Association between colour of liquor and asphyxia

Colour of Liquor	Asphyxia -	%	Asphyxia +	%
Clear	237	86.50	17	36.96
Meconium Stained	37	13.50	29	63.04
Total	274	100	46	100

Colour of Liquor	Clear	%	Meconium Stained	%
No Asphyxia	237	93.31	37	56.06
Asphyxia +	17	6.69	29	43.94
Total	254	100	66	100

Chi-squared Test	
chi-squared statistic	5.90
Degrees of Freedom	1
P value	0.968



By conventional criteria the association between the Birth Asphyxia and colour of liquor is considered to be statistically not significant since $p > 0.05$.

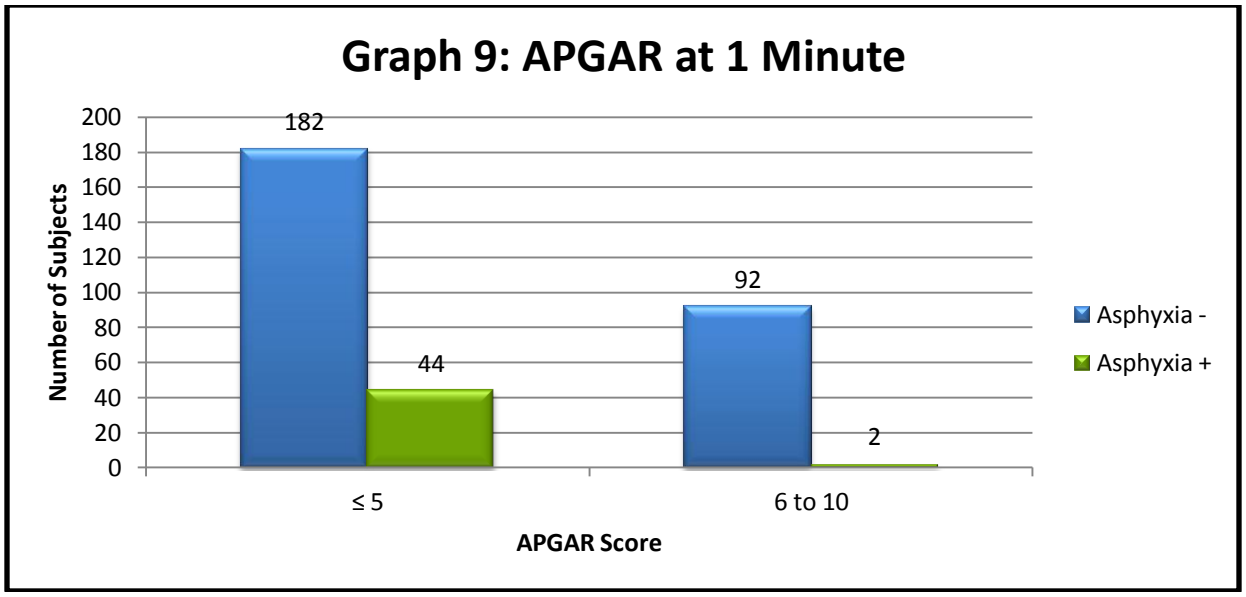
APGAR @ 1 MIN

Table 24: Association between asphyxia and APGAR score at 1st minute

APGAR @ 1 MIN	No Asphyxia	%	Asphyxia +	%
≤ 5	182	66.42	36	78.26
6 to 10	92	33.58	10	21.74
Total	274	100	46	100

APGAR @ 1 MIN	≤ 5	%	6 to 10	%
No Asphyxia	182	80.53	92	97.87
Asphyxia +	44	19.47	2	2.13
Total	226	100	94	100

APGAR @ 1 MIN	No Asphyxia	Asphyxia +
N	274	46
Mean	5.635036496	3.065217
SD	0.993575793	1.4667
P value Unpaired t - test	0.0000*	



By conventional criteria the association between the birth asphyxia and APGAR scores at 1 minute is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among the groups and the difference is significant. In simple terms, in emergency LSCS, the incidence of birth asphyxia is more when the APGAR scores at 1 minute decreased. It is statistically significant with a p-value of 0.0000 according to unpaired t-test.

Clinical Significance

The average and APGAR scores at 1 minute was 3.07±1.47 in Asphyxia+ group compared to 5.64±0.99 in No Asphyxia- group.

The APGAR scores at 1 minute decreased was meaningfully less in the Asphyxia+ group compared to No Asphyxia- group by 2.57 points.

The occurrence of birth asphyxia was meaningfully more (78.26%).when the APGAR scores at 1 minute decreased below 5.

This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is real intrapartum obstetric risk factor for developing birth asphyxia if the APGAR scores at 1 minute is significantly low.

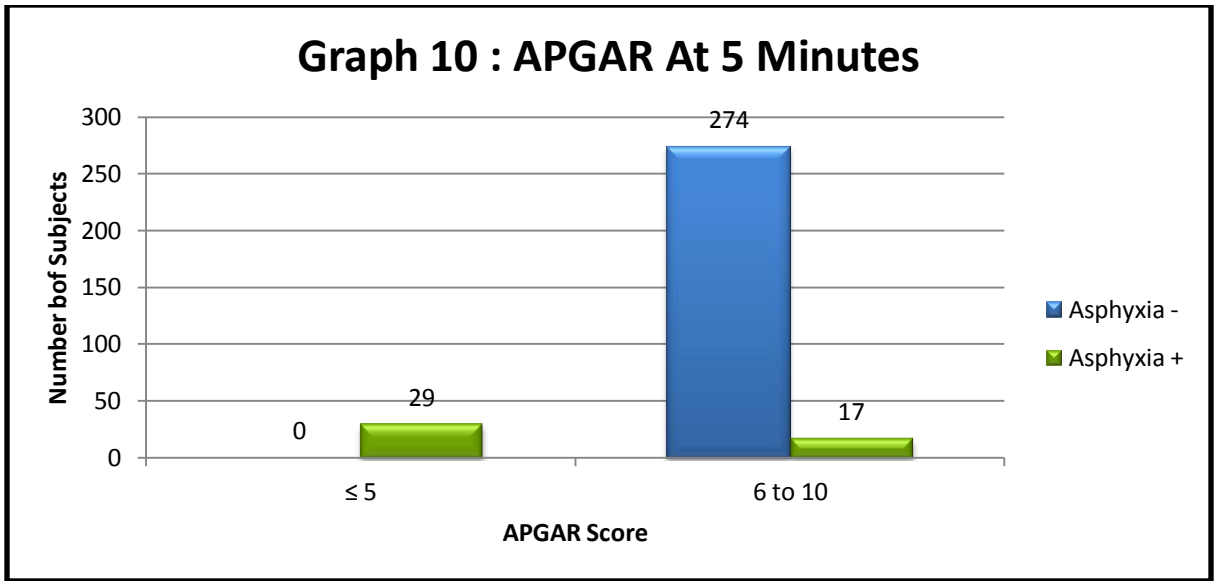
APGAR @ 5 MIN

Table 25 : Association between asphyxia and APGAR score at 5th minute

APGAR @ 5 MIN	No Asphyxia	%	Asphyxia +	%
≤ 5	0	0.00	29	63.04
6 to 10	274	100.00	17	36.96
Total	274	100	46	100

APGAR @ 5 MIN	≤ 5	%	6 to 10	%
No Asphyxia	0	0.00	274	94.16
Asphyxia +	29	100.00	17	5.84
Total	29	100	291	100

APGAR @ 5 MIN	No Asphyxia	Asphyxia +
N	274	46
Mean	7.386861	5.521739
SD	0.643345	1.110338
P value Unpaired t - test	0.0000*	



By conventional criteria the association between the birth asphyxia and APGAR scores at 5 minute is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among the groups and the difference is significant. In simple terms, in emergency LSCS, the incidence of birth asphyxia is more when the APGAR scores at 5 minute decreased. It is statistically significant with a p-value of 0.0000 according to unpaired t-test.

Clinical Significance

The average and APGAR scores at 5 minute was 5.52±1.11 in Asphyxia+ group compared to 7.39±0.64 in No Asphyxia group.

The APGAR scores at 5 minute decreased was meaningfully less in the Asphyxia+ group compared to No Asphyxia group by 1.87 points.

The occurrence of birth asphyxia was meaningfully more (63.04%).when the APGAR scores at 5 minute decreased below 5.

This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that there is real intrapartum obstetric risk factor for developing birth asphyxia if the APGAR scores at 5 minute is significantly low

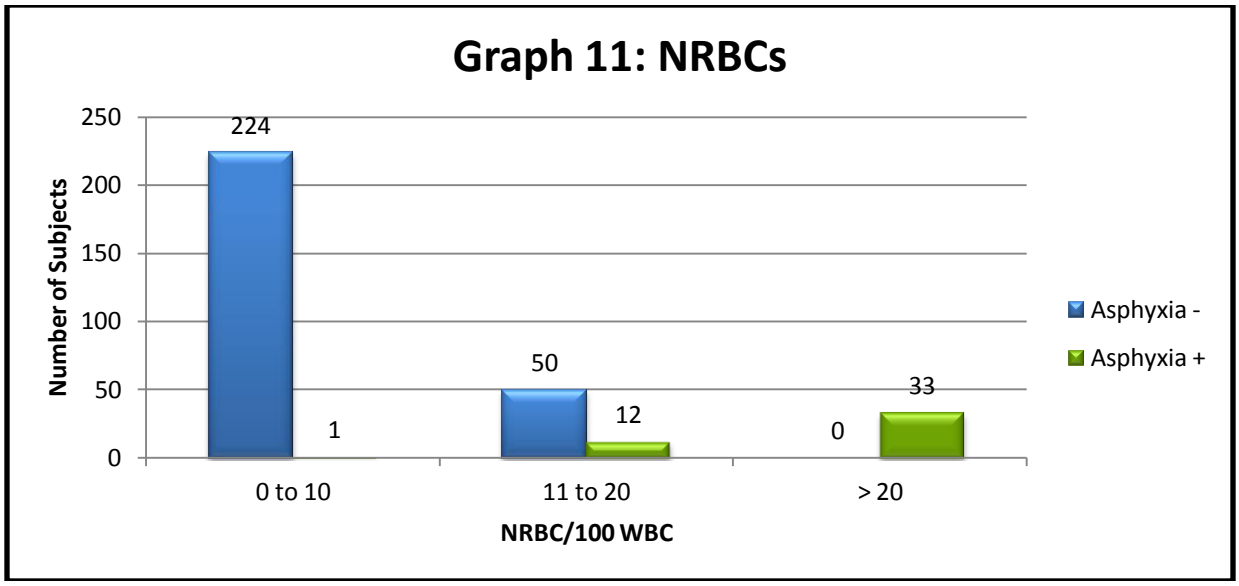
NRBCs

Table 26 : Tabulation showing relation between NRBCS and Asphyxia

NRBCs	No Asphyxia	%	Asphyxia +	%
0 to 10	224	81.75	1	2.17
11 to 20	50	18.25	12	26.09
> 20	0	0.00	33	71.74
Total	274	100	46	100

NRBCs	0 to 10	%	11 to 20	%	> 20	%
No Asphyxia	224	99.56	50	80.65	0	0.00
Asphyxia +	1	0.44	12	19.35	33	100.00
Total	225	100	62	100	33	100

	No Asphyxia	Asphyxia +
N	274	46
Mean	8.040146	25.36957
SD	2.663385	9.367458
P value Unpaired t - test	0.000*	



By conventional criteria the association between the birth asphyxia and elevated NRBCs is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among the groups and the difference is significant. In simple terms, in emergency LSCS, the incidence of birth asphyxia is more when the NRBCs levels are significantly elevated. It is statistically significant with a p-value of 0.000 according to unpaired t-test.

Clinical Significance

The average NRBCs level was 25.37±9.37 NRBCs per 100 WBC in Asphyxia+ group compared to 8.04±2.66 NRBCs per 100 WBC in No Asphyxia group.

The NRBCs level increased meaningfully more in the Asphyxia+ group compared to No Asphyxia group by 17.33 NRBCs per 100 WBC.

The occurrence of birth asphyxia was meaningfully more (97.83%).when the NRBCs level increased more than 10 NRBCs per 100 WBC

This difference is true and significant and has not occurred by chance.

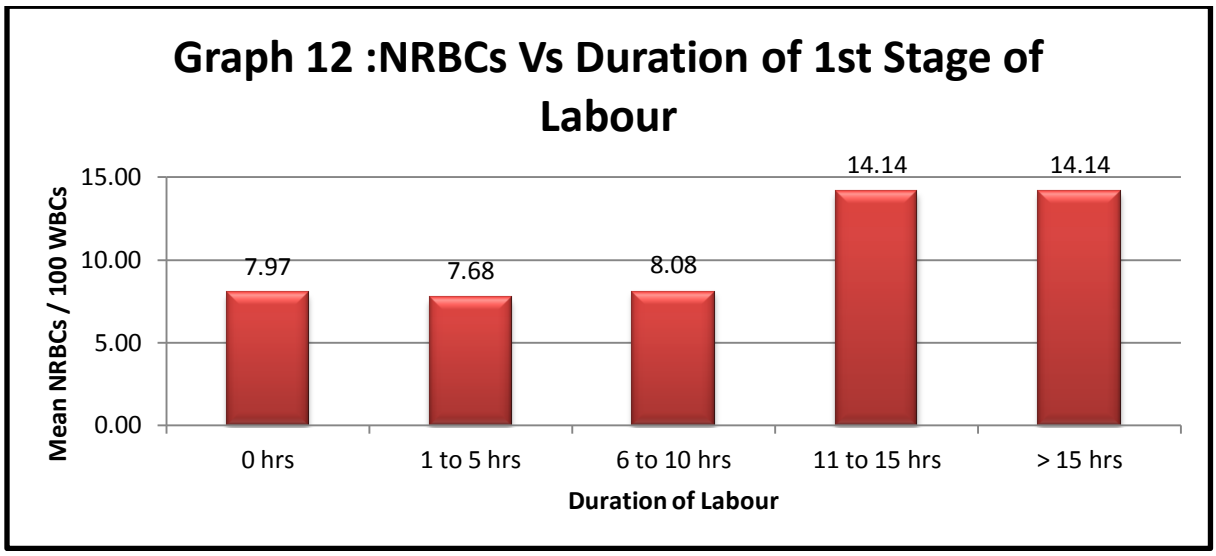
Conclusion

We conclude that increased NRBCs levels correlates well with development of birth asphyxia, and can be used as a simple and reliable index for assessment of severity and early outcome of perinatal asphyxia.

NRBCs Vs Duration of 1st Stage of Labour

Table 27: NRBC tabulated based on the duration of 1st stage of labour

NRBCs	0 hrs	1 to 5 hrs	6 to 10 hrs	11 to 15 hrs	> 15 hrs
N	139	22	38	115	6
Mean	7.97	7.68	8.08	14.14	14.14
SD	2.63	2.71	2.53	10.08	10.08
P value Unpaired t test		0.02			



By conventional criteria the association between the NRBCs levels and duration of 1st stage of labour is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among the groups and the difference is significant. In simple terms, in emergency LSCS, the NRBCs level is more when

the duration of 2nd stage of labour increases. It is statistically significant with a p-value of 0.02997 according to unpaired t-test.

Clinical Significance

The average NRBCs levels during the duration of 2nd stage of labour varied from 9.48±5.55 per 100 WBCs(0Hours) to 13.57±10.72 per 100 WBCs(> 2 Hours). The occurrence of higher NRBCs levels was meaningfully more (1.43 times) when the duration of 2nd stage of labour increased more than 1 hour with a mean increase of 4.09 NRBCs per 100 WBCs. This difference is true and significant and has not occurred by chance. .

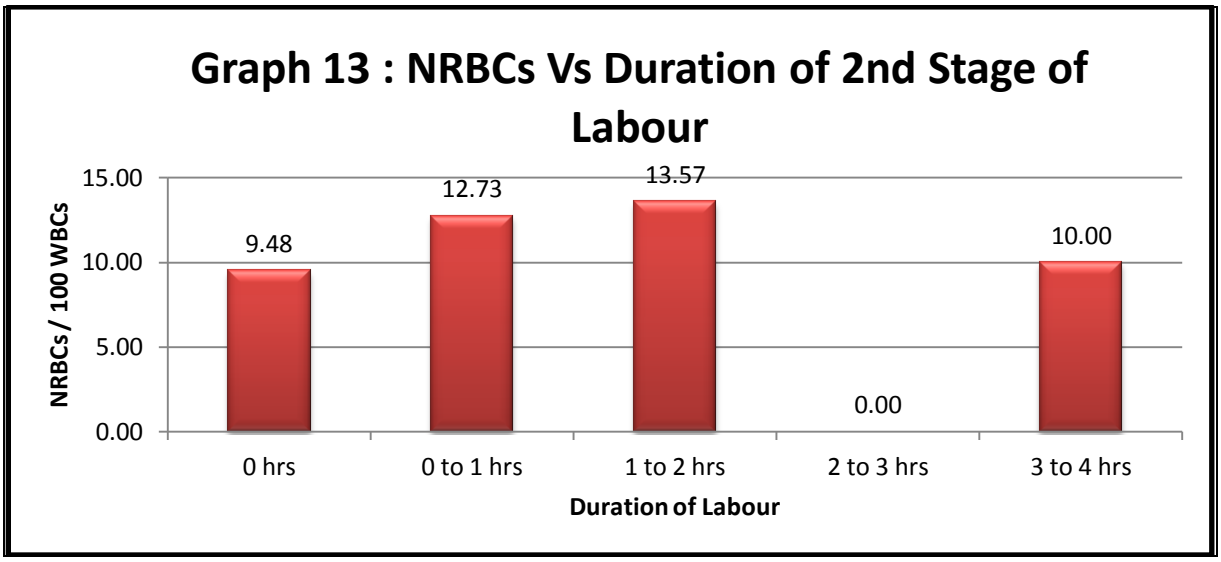
Conclusion

We conclude that there is real intrapartum obstetric risk factor for developing birth asphyxia if the duration of 2nd stage of labour exceeds 1 hour which is also substantiated by the corresponding increase in NRBCs level. Thus NRBCs level can be used as a surrogate predictor of birth asphyxia in prolonged 1st stage of labour.

NRBCs Vs Duration of 2nd Stage of Labour

Table 28: NRBC tabulated based on the duration of 2nd stage of labour

NRBCs	0 hrs	0 to 1 hrs	1 to 2 hrs	2 to 3 hrs	3 to 4 hrs
N	231	26	61	0	2
Mean	9.48	12.73	13.57	0.00	10.00
SD	5.55	10.57	10.72	0.00	0.00
P value Unpaired t test		0.02997			



By conventional criteria the association between the NRBCs levels and duration of 2nd stage of labour is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among the groups and the difference is significant. In simple terms, in emergency LSCS, the NRBCs level is more when the duration of 2nd stage of labour increases. It is statistically significant with a p-value of 0.02997 according to unpaired t-test.

Clinical Significance

The average NRBCs levels during the duration of 2nd stage of labour varied from 9.48±5.55 per 100 WBCs (0Hours) to 13.57±10.72 per 100 WBCs(> 2 Hours). The occurrence of higher NRBCs levels was meaningfully more (1.43 times) when the duration of 2^{nd} stage of labour increased more than 1 hour with a mean increase of 4.09 NRBCs per 100 WBCs. This difference is true and significant and has not occurred by chance. .

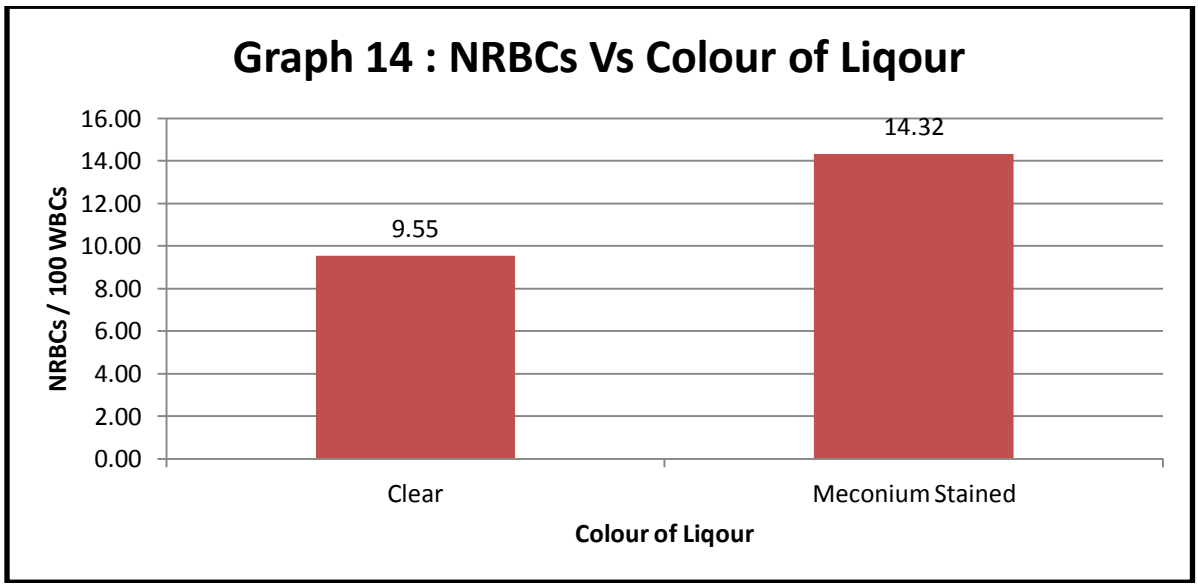
Conclusion

We conclude that there is real intrapartum obstetric risk factor for developing birth asphyxia if the duration of 2^{nd} stage of labour exceeds 1 hour which is also substantiated by the corresponding increase in NRBCs level. Thus NRBCs level can be used as a surrogate predictor of birth asphyxia in prolonged 1^{st} stage of labour.

NRBCs Vs Colour of Liquor

Table 29: Mean NRBC count with Meconium Stained

NRBCs	Clear	Meconium Stained
N	254	66
Mean	9.55	14.32
SD	6.66	9.03
P value Unpaired t test	0.1881	



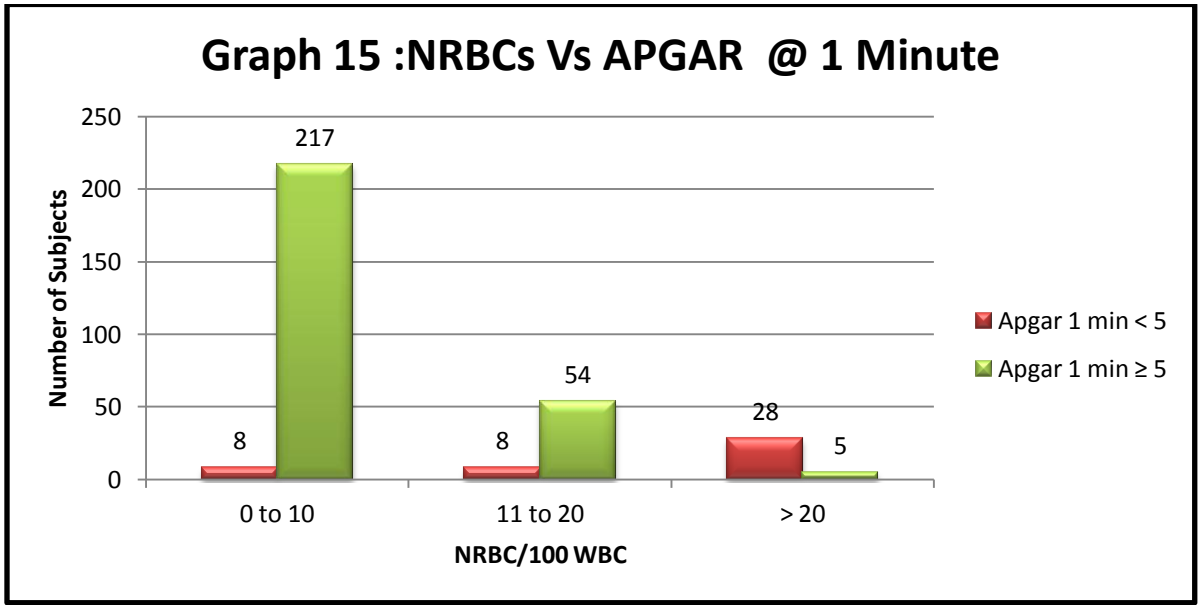
By conventional criteria the association between the NRBCs levels and colour of liquor is considered to be not statistically significant since $p > 0.05$.

NRBCs Vs APGAR @ 1 Minute

Table 30 : NRBC tabulated along with the APGAR at 1 minute

NRBCs	Apgar @ 1 min < 5	%	Apgar @1 min ≥ 5	%
0 to 10	8	18.18	217	78.62
11 to 20	8	18.18	54	19.57
> 20	28	63.64	5	1.81
Total	36	100	276	100.00

NRBCs	Apgar @ 1 min < 5	Apgar @ 1 min ≥ 5
N	44	276
Mean	23.64	8.44
SD	11.59	3.40
t test	0.0004	



By conventional criteria the association between the NRBCs level and APGAR scores at 1 minute is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among the groups and the difference is significant. In simple terms, in emergency LSCS, the levels of NRBCs is more when the Apgar scores at 1 minute decreased. It is statistically significant with a p-value of 0.0004 according to unpaired t-test.

Clinical Significance

The average NRBCs per 100 WBC when matched with Apgar scores at 1 minute was 23.64±11.59 in Apgar @ 1 min < 5 group compared to 8.44±3.40 in Apgar @ 1 min ≥ 5 group.

The average NRBCs per 100 WBC when matched with Apgar scores at 1 minute was was meaningfully less in Apgar @ 1 min < 5 group compared to Apgar @ 1 min ≥ 5 group by 15.19 NRBCs per 100 WBC.

The increase of NRBCs per 100 WBC was meaningfully 2.80 times more when the Apgar scores at 1 minute decreased below 5.

This difference is true and significant and has not occurred by chance.

Conclusion

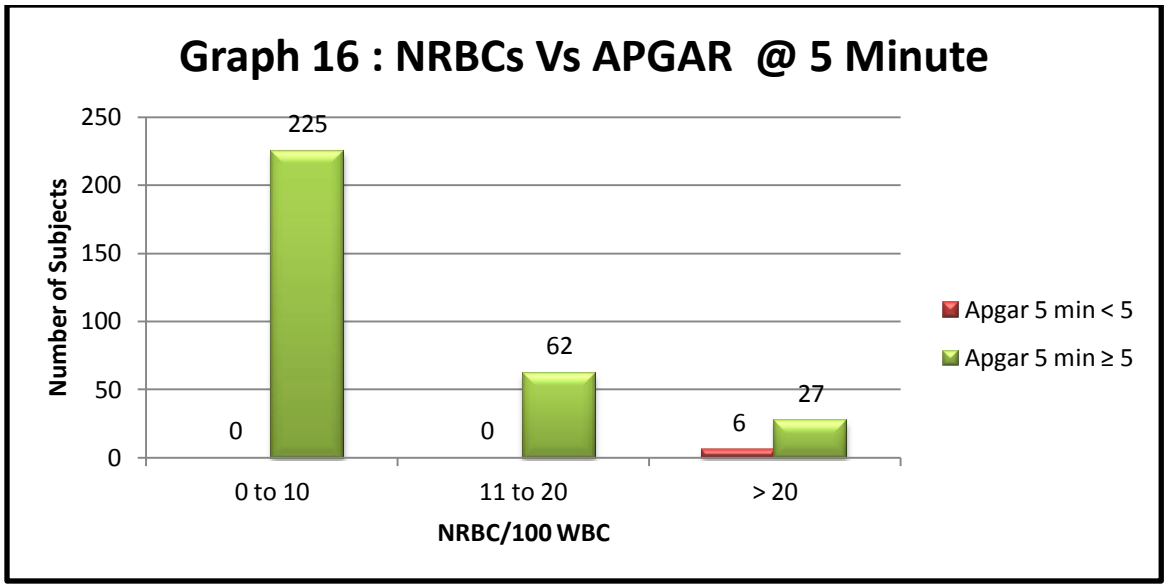
We conclude that there is real intrapartum obstetric risk factor for developing high levels of NRBCs per 100 WBC if the APGAR scores at 1 minute is significantly low.

NRBCs Vs APGAR @ 5 Minute

Table 31 : NRBC tabulated along with the APGAR at 5 minute

NRBCs	Apgar @ 5 min < 5	%	Apgar @ 5 min ≥ 5	%
0 to 10	0	0.00	225	71.66
11 to 20	0	0.00	62	19.75
> 20	6	100.00	27	8.60
Total	6	100	314	100.00

NRBCs	Apgar @ 5 min < 5	Apgar @ 5 min ≥ 5
N	6	314
Mean	30.67	10.15
SD	2.07	6.97
t test	0.0013	



By conventional criteria the association between the NRBCs level and APGAR scores at 5 minute is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among the groups and the difference is significant. In simple terms, in emergency LSCS, the levels of NRBCs is more when the Apgar scores at 5 minute decreased. It is statistically significant with a p-value of 0.0013 according to unpaired t-test.

Clinical Significance

The average NRBCs per 100 WBC when matched with Apgar scores at 5 minute was 30.67±2.07 in Apgar @ 5 min < 5 group compared to 10.15±6.97 in Apgar @ 5 min ≥ 5 group.

The average NRBCs per 100 WBC when matched with Apgar scores at 5 minute

was was meaningfully less in Apgar @ 5 min < 5 group compared to Apgar @ 5 min ≥ 5 group by 20.52 NRBCs per 100 WBC.

The increase of NRBCs per 100 WBC was meaningfully 3.02 times more when the APGAR scores at 5 minute decreased below 5.

This difference is true and significant and has not occurred by chance.

Conclusion

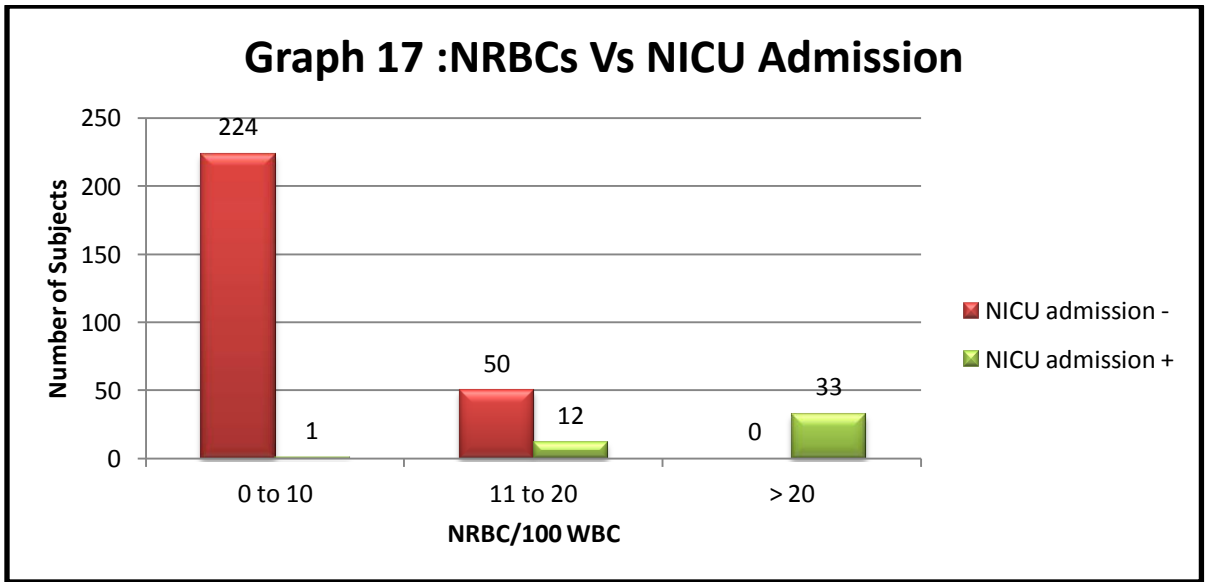
We conclude that there is real intrapartum obstetric risk factor for developing high levels of NRBCs per 100 WBC if the APGAR scores at 5 minute is significantly low.

NRBCs Vs NICU Admission

Table 32: NICU admission and its relation to NRBC

NRBCs	NO NICU admission	%	NICU admission	%
0 to 10	224	81.75	1	2.17
11 to 20	50	18.25	12	26.09
> 20	0	0.00	33	71.74
Total	274	100	46	100

NRBCs	NO NICU admission	NICU admission
N	274	46
Mean	8.040146	25.36957
SD	2.663385	9.367458
t test	0.0000	

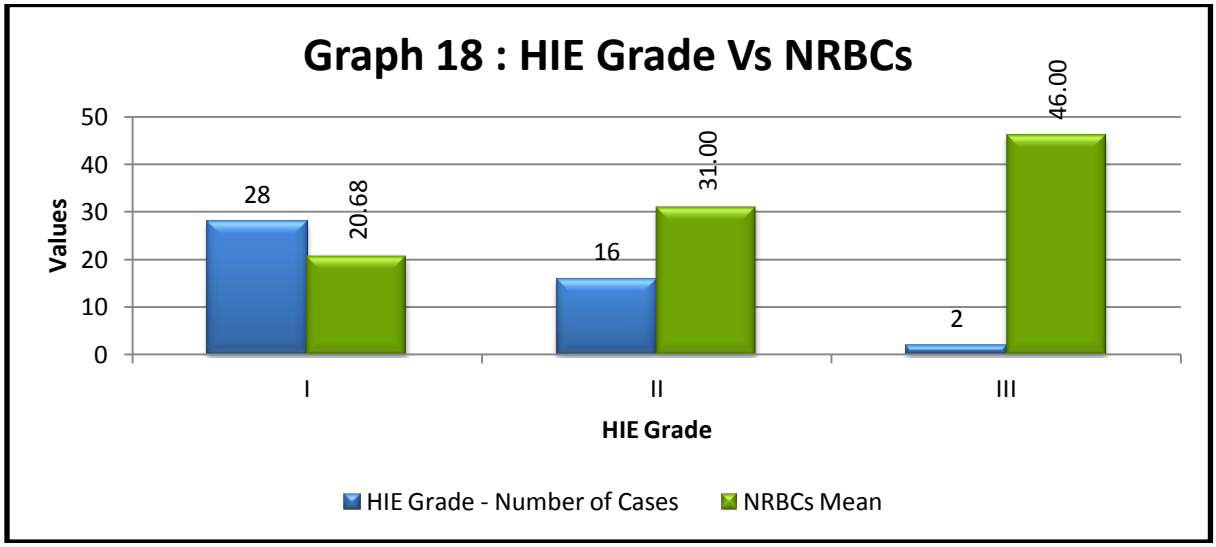


HIE Vs NRBCs

Table 33 : Relation between NRBC and HIE grading

HIE Grade	No of Cases	NRBCs Mean±SD
I	28	20.68±7.32
II	16	31.00±5.93
III	2	46.00±0.00

	No of cases	NRBCs
N	46	46
Mean	1.434783	25.36957
SD	0.583178	9.367458
P value Unpaired t - test	0.000*	



By conventional criteria the association between the HIE grading and elevated NRBCs is considered to be statistically significant since $p < 0.05$.

Statistical Significance

This indicates that there is a true difference among the groups and the difference is significant. In simple terms, in emergency LSCS, significantly higher NRBCs level was found in those who developed hypoxic ischemic encephalopathy in early neonatal period. It is statistically significant with a p-value of 0.000 according to unpaired t-test.

Clinical Significance

The average NRBCs level was 20.68±7.32 NRBCs per 100 WBC in HIE Grade 1, 31.00±5.93 NRBCs per 100 WBC in HIE Grade 2 and 46.00±0.00 NRBCs per 100 WBC in HIE Grade 3.

The NRBCs level increased meaningfully more in the HIE group 2 compared to HIE group 1 by 10.32 NRBCs per 100 WBC.

The NRBCs level increased meaningfully more in the HIE group 3 compared to HIE group 2 by 15.00 NRBCs per 100 WBC.

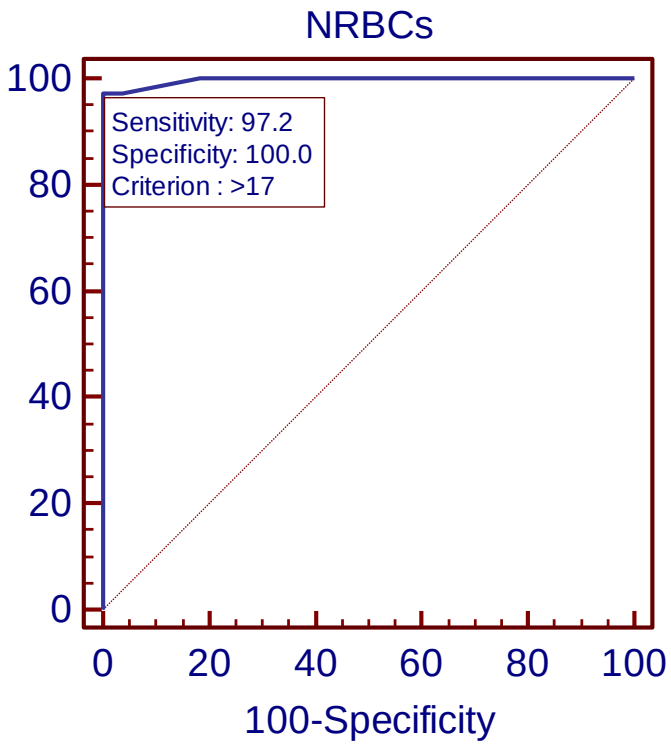
The NRBCs level increased meaningfully more in the HIE group 3 compared to HIE group 1 by 25.32 NRBCs per 100 WBC.

The levels of NRBCs in HIE Grade 3 was meaningfully more (145%) when compared to NRBCs level in HIE Grade 2 and also meaningfully more (69%) when compared to NRBCs level in HIE Grade 2.

This difference is true and significant and has not occurred by chance.

Conclusion

We conclude that increased NRBCs levels correlates well with development of birth asphyxia. Hence NRBC levels can be a useful for the evaluation of perinatal asphyxia where facilities of pH sampling are not available and can serve as a reliable, inexpensive and easily available marker of perinatal asphyxia.



ROC curve

Variable	NRBCs
Classification variable	HIE_grade HIE grade

Sample size		320
Positive group :	HIE grade = 1	36
Negative group :	HIE grade = 0	284

Disease prevalence (%)	unknown

Area under the ROC curve (AUC)

Area under the ROC curve (AUC)	0.996919
Standard Error[a]	0.00311
95% Confidence interval[b]	0.982788 to 0.999925
z statistic	159.857
Significance level P (Area=0.5)	<0.0001

[a] DeLong et al., 1988
[b] Binomial exact

Youden index

Youden index J	0.9722
Associated criterion	>17

Criterion values and coordinates of the ROC curve

Criterion	Sensitivity	95% CI	Specificity	95% CI	+LR	-LR
≥2	100.00	90.3 - 100.0	0.00	0.0 - 1.3	1.00	
>11	100.00	90.3 - 100.0	81.69	76.7 - 86.0	5.46	0.00
>12	97.22	85.5 - 99.9	96.13	93.2 - 98.1	25.10	0.029
>17	97.22	85.5 - 99.9	100.00	98.7 - 100.0		0.028
>46	0.00	0.0 - 9.7	100.00	98.7 - 100.0		1.00

ROC curve shows high sensitivity to detect birth asphyxia by nucleated RBC’s .

DISCUSSION

In the present study, cord blood collected from 320 patients singleton term pregnancies, primi or multi, babies of more than 2.5 kg , appropriate for gestational age delivered by emergency LSCS irrespective of indication without any maternal co -morbid factors. Nucleated RBC/100 WBC count was measured in the cord blood collected.

The basic definition of perinatal asphyxia was based on an umbilical arterial pH of ≤ 7.15 along with an Apgar score of < 6/10 at 5minutes of birth.

Apgar Score:

In present study, an Apgar score of < 6/10 at 5 minutes was necessary to define perinatal asphyxia. This cut off value for Apgar score is in keeping with the definition of moderate birth asphyxia (Nelson et al., 1996) requiring active neonatal resuscitation and is currently accepted world wide.

The Apgar score at 5 minutes, which has been correlated with long term neurologic impairment (Nelson and Jonas, 1981) rather than immediate neonatal status in the past, noted in the present study in all newborns, was taken into consideration to define perinatal asphyxia.

In previous studies, however, different cut off values for Apgar score have been taken to define birth asphyxia, which adds to the ambiguity associated with

the Apgar Score, Sykes et al (1982) took a value of < 7/10 at both one minute and 5 minutes to define asphyxia, whereas Gilstrap et al (1989) considered as Apgar score at one and 5 minutes of ≤ 3/10 to be indicative of significant birth asphyxia.

In the past studies have also reported on the poor correlation of Apgar score with biochemical abnormalities in cord blood (Sykes et al, 1982; Silverman et al, 1985).

The present study did not aim at studying the relationship between Apgar score and cord blood gas analysis.

Correlation of passage of meconium with Apgar score and umbilical artery pH:

Low (1988) had reported poor correlation between fetal acidosis, Apgar score and presence of meconium in a study conducted on 1773 patients. The false positive rates of meconium in the prediction of fetal acidosis were 95% and of Apgar score 0-3 at one minute was 84%. He also reported a low sensitivity of Apgar score and meconium in predicting of fetal asphyxia. In another similar study by Abramovici et al., 1974 the authors reported no significant difference in terms of fetal pH, Apgar score and perinatal outcome in those with meconium and in those in whom amniotic fluid was clear.

Starks (1980) in a comparative study comprising of 177 patients with meconium staining and 100 patients without meconium, concluded that patients

with thick meconium had significantly lower one and five minutes Apgar scores and scalp pH as compared to thin meconium and no meconium groups.

Nucleated RBC/100 WBC as a marker of perinatal asphyxia:

1) Normal value of nucleated RBC/100 WBC in umbilical venous cord blood in term non-asphyxiated newborns:

In the present study, the number of nucleated red blood cells/100 white blood cells in the asphyxia group is high .

The incidence of birth asphyxia is more when the NRBCs levels are significantly elevated. It is statistically significant with a p-value of 0.000 according to unpaired t-test.

The average NRBCs level was 25.37±9.37 NRBCs per 100 WBC in Asphyxia+ group compared to 8.04±2.66 NRBCs per 100 WBC in No Asphyxia group.

The NRBCs level increased meaningfully more in the Asphyxia+ group compared to No Asphyxia group by 17.33 NRBCs per 100 WBC.

The occurrence of birth asphyxia was meaningfully more (97.83%).when the NRBCs level increased more than 10 NRBCs per 100 WBC

Table 34 : Showing Normal values of nucleated RBCs/100 WBC in umbilical venous blood in different studies

Authors	**No.Of patients**	**Mean nucleated RBC'S/100 WBC'S**	**S.D**
Sinha et at (1972)	84	2.3	0.69
Shiv Hare et al (1976)	33	4.1	2.4
Green & Mimouni (1990)	102	1.7	6.2
Phelan et al (1995)	83	3.4	3.0
Hanlon Lundberg et al (1997)	1112	8.55	10.27
Hanlon Lundberg & Kirby(1999)	1561	9.2	18.1
Desai papa et al (2008)	51	12.53	5.51
Present study	274	8.04	2.66

2) Effect of maternal age on the number of nucleated RBC / 100 WBC in umbilical venous sample:

All mothers in our study population had no significant correlation with there age and number of NRBC's

By conventional criteria the association between the Birth Asphyxia and age is

considered to be not statistically significant since $p > 0.05$.

Since age is not statistically significant, it means that there is no difference between the groups. In other words the groups contain subjects with the same basic demographic characteristics.

3) Effect of gravida on the number of nucleated RBC/100 WBC in umbilical venous sample:

All mothers in our study population had no significant correlation with there gravidity and number of NRBC's .

By conventional criteria the association between the Birth Asphyxia and Gravida is considered to be not statistically significant since $p > 0.05$.

4) Effect of Indication of Emergency LSCS and number of nucleated RBC/100 WBC in umbilical venous sample:

In the present study there exists an association between the Birth Asphyxia and Indication for Emergency LSCS , and is considered to be statistically significant.

In emergency LSCS, the incidence of birth asphyxia is more when there is a Fetal Distress and Deep transverse arrest compared to other indications for emergency LSCS. It is statistically significant with a p-value of 0.000 according to Chi-squared Test.

The occurrence of birth asphyxia was meaningfully more (35%) when the indication for emergency LSCS was Fetal Distress and Deep transverse arrest (46.6%) compared to other indications like failed induction (17.39%) and prev LSCS.

5) Association of nucleated RBC/100 WBC in umbilical venous sample with duration of labour:

In the present study there exists an association between birth asphyxia and duration of labour, and is considered to be stastistically significant.

In emergency LSCS, the incidence of birth asphyxia is more when the duration of labour increases.

The average duration of 1^{st} stage of labour was 9.83±5.43 hours in Asphyxia+ group compared to 5.42±2.86 hours in No Asphyxia group. The occurrence of birth asphyxia was meaningfully more (67.39%).when the the duration of 1^{st} stage of labour increased more than 10 hours. The duration of 1^{st} stage of labour was meaningfully more (81%) in the Asphyxia+ group compared to No Asphyxia group by 4 hours and 24 minutes.

The average duration of 2^{nd} stage of labour was 2.35±0.85 hours in Asphyxia+ group compared to 1.45±0.84 hours in No Asphyxia- group. The occurrence of birth asphyxia was meaningfully more (58.70%).when the the duration of 2^{nd} stage

of labour increased more than 2 hours. The duration of 2nd stage of labour was meaningfully more (62%) in the Asphyxia+ group compared to No Asphyxia group by 54 minutes.

The real intrapartum obstetric risk factor for developing Birth asphyxia if the duration of 1st stage of labour exceeds 10 hours and 2nd stage exceeds 2 hours.

6) Association of nucleated RBC/100 WBC in umbilical venous sample with Meconium staining of liquor:

Hanlon Lundberg et al in two different studies (1997 and 1999) had concluded that an elevated nucleated RBC level was seen in new borns with meconium stained liquor since the presence of meconium indicated some form of fetal distress which could trigger off a reactionary erythroblastosis in the fetus. In the present study, however, no significant correlation could be demonstrated between presence of meconium and number of nucleated RBC/100 WBC .

7). Association of nucleated RBC/100 WBC in umbilical venous sample with Apgar score:

In the present study, a significant correlation was found between Apgar score and number of nucleated RBC/100 WBC with higher number of nucleated RBCs seen in those with lower Apgar score. Contradictory reports are available

regarding correlation of nucleated RBC with Apgar score. Thilaganathan et al., (1994) refuted the assumption that higher nucleated RBC levels are seen in new born with lower Apgar Scores. In 1997 however, Hanlon Lundberg et al., demonstrated significantly higher levels of nucleated RBCs in newborns with Apgar Score of 0-3 than those in with a score between 7 and 10.

8) Association of nucleated RBC / 100 WBC in umbilical venous sample with fetal heart variability:

By conventional criteria the association between the Birth Asphyxia and foetal heart variability.

In present study the incidence of birth asphyxia is more when there is a foetal heart variability compared to absence of it.

The occurrence of birth asphyxia was meaningfully more (52.17%) when there is presence of foetal heart rate variability compared to its absence (47.83%).

9) Association of nucleated RBC / 100 WBC in umbilical venous sample with adverse neonatal outcome:

In the present study a significant higher level of nucleated RBC/100 WBC in umbilical venous blood was seen at birth in new born who develop hypoxic ischemic encephalopathy in the early neonatal period.

This finding is consistent with previous studies (Phelan et al., 1995, Korst et al., 1996) which showed higher nucleated RBC levels in neonates with neurological impairement. Korst et al., (1996) compared the maximum level of nucleated RBC and also the clearance time in addition to measuring initial levels of nucleated RBC/100 WBCs. Such analysis was not included in the present study.

In this study significantly higher NRBCs level was found in those who developed hypoxic ischaemic encephalopathy in early neonatal period. It is statistically significant with a p-value of 0.000 according to unpaired t-test.

The average NRBCs level was 20.68±7.32 NRBCs per 100 WBC in HIE Grade 1, 31.00±5.93 NRBCs per 100 WBC in HIE Grade 2 and 46.00±0.00 NRBCs per 100 WBC in HIE Grade 3.

The NRBCs level increased meaningfully more in the HIE group 2 compared to HIE group 1by 10.32 NRBCs per 100 WBC.

The NRBCs level increased meaningfully more in the HIE group 3 compared to HIE group 2 by 15.00 NRBCs per 100 WBC.

The NRBCs level increased meaningfully more in the HIE group 3 compared to HIE group 1 by 25.32 NRBCs per 100 WBC.

The levels of NRBCs in HIE Grade 3 was meaningfully more (145%) when compared to NRBCs level in HIE Grade 2 and also meaningfully more (69%) when compared to NRBCs level in HIE Grade 2.

SUMMARY

1. In the present study cord blood have been collected from 320 patients Singleton term pregnancies primi /multi babies of more than 2.5kg appropriate for gestational age delivered by emergency lscs irrespective of indication without any maternal co morbid factors.

2. Nucleated RBC'S have been estimated from the sample

3. Mean ± SD of mothers of newborn was 27.839 in 274 non asphyxiated groups. The associated between the birth asphyxia and age is considered to be not statistically significant since p >0.05

4. In the study population, 133 (41.56 %) belong to primigravida category and the rest, 187 (58.44%) belong to multigravida category. The association between the Birth Asphyxia and Gravida is not statistically significant (p > 0.05).

5. In present study majority of the women 85 (26.56%) had previous LSCS as an indication for emergency LSCS followed by failed induction 71 (22.19%) and fetal distress 60 (18.75%) and DTA15(4.69%). Among the birth asphyxia group, the major indication for emergency LSCS was fetal distress with 45.65% and DTA 46.6% babies are asphyxiated and it was found to be statistically significant.

6. Most of the study participants, 139 (43.44%) did not have delay in the first stage of labour, but 35.94% of the women had 11 – 15 hours of delay during the first stage of labour. The mean duration of first stage of labour was 9.83 hours in birth asphyxia group and 5.44 hours in no birth asphyxia group. The mean duration was higher in birth asphyxia group and it was found to be statistically significant ($p < 0.05$).The average duration of 2nd stage of labour was 2.35±0.85 hours in Asphyxia+ group compared to 1.45±0.84 hours in No asphyxia group. It is statistically significant with p value of 0.01598.

7. Among the birth asphyxia group, fetal heart rate variability was found in 54.17% compared to 21.90% among no birth asphyxia group, which is statistically significant $p< 0.05$.

8. Among the study participants, 254 (79.38%) had clear liquor, while 20.62% had meconium stained liquor. In the birth asphyxia group, higher percentage (63.04%) had meconium stained liquor than the other group, but the association was not found to be statistically significant ($P> 0.05$).

9. Among the study participants, the mean apgar score at 1 minute was 3.07 $\pm$ 1.47 in birth asphyxiated babies. It was less when compared to no birth asphyxia group's apgar score at 1 minute 5.64 $\pm$ 0.99. The mean apgar score at 5 minutes was 5.52 $\pm$ 1.11 in birth asphyxiated babies. It was less when compared to no birth asphyxia group's apgar score at 5 minutes 7.39 $\pm$ 0.64.

About 63.04% in the asphyxia group had apgar score < 5, whereas no one in the other group had apgar score < 5. Those babies with less APGAR score at one minute and 5 minutes had more birth asphyxia and this association was found to be statistically significant ($p<0.05$).

10. Among the study group, 225 (70.31%) had NRBC < 10, whereas 29.69% had NRBC count > 10. The mean NRBC count was much higher among birth asphyxia group [25.37 $\pm$ 9.37], compared to no birth asphyxia group [8.04 $\pm$ 2.66] and this association was found to be statistically significant ($P<0.05$).
11. The average NRBCs level was 20.68±7.32 NRBCs per 100 WBC in HIE Grade 1, 31.00±5.93 NRBCs per 100 WBC in HIE Grade 2 and 46.00±0.00 NRBCs per 100 WBC in HIE Grade 3.
12. We conclude that increased NRBCs levels correlates well with development Birth asphyxia. Hence NRBC levels can be a useful for the evaluation of perinatal asphyxia where facilities of pH sampling are not available and can serve as a reliable, inexpensive and easily available marker of perinatal asphyxia.
13. Neonate with Nucleated RBC'S more than 10 was admitted to NICU and had poor neonatal outcome. Estimation of NRBCs may be an easy and simple investigation and may be used as an indicator of fetal asphyxia in the future.

CONCLUSION

Present prospective crossectional study on prediction of perinatal asphyxia by the presence of nucleated RBCs/100 WBCs provided evidence that higher nucleated RBC/100 WBCs count was seen in umbilical cord venous blood sample in new born with acute intrapartum asphyxia (as evidence by Lower Apgar Score). Higher nucleated RBC/100 WBC in umbilical venous sample was also correlated with poor, early neonatal outcome i.e., neonatal NICU admission.

However, no correlation could be demonstrated between the number of nucleated RBC/100 WBC and neurological development of baby till 16 weeks of age.

- This study proved that meconium stained liquor per se, is not the cause/predictor/ indicator of intrapartum asphyxia.
- Low Apgar score at 5 minutes of life denote chronic asphyxia and definitely can be considered as clinical marker of asphyxia.
- However, this study lacked the corroboration of these facts with measurement of umbilical venous pH or fetal blood gas analysis, postnatally.
- Low Apgar scores together with umbilical venous pH and arterial blood gas analysis can most definitely be marker of asphyxia and predictor of adverse neonatal outcomes.

To conclude estimating the number of nucleated RBC/100 WBC in umbilical cord venous blood sample of new born is an important test, the sample being obtained non invasively from otherwise discarded specimen and analyzed by personnel or equipment readily available in most hospital laboratories. The level of nucleated RBCs/100 WBCs correlates with acute intrapartum asphyxia and can be used as an index of early neonatal outcome.

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PROFORMA

NAME: AGE: IP NO:

ADDRESS:

OBSTETRIC HISTORY:

1. Obstetric score
2. Regular ANC
3. LMP EDD
4. Menstrual history: Regular/Irregular
5. Marital history: Married since : Consanguinity:
6. Previous OBS history: Gravia- Para- Live- Abortion- Dead/Still born

   Normal / Instrumental- Preterm/ Term-

   LSCS: Term/ Preterm Indications:

   Complications: Antepartum/ Intrapartum / Postpartum

MEDICAL HISTORY:

1. Hypertension/ Jaundice/ Tuberculosis/ Heart disease/ Epilepsy/ Drug intake/ Diabetes mellitus/ Bronchial asthma/ Thyroid disorder/ Surgeries/ Others

PRESENT PREGNANCY:

1. Confirmation of pregnancy: When / Where
2. Booked and Immunized:
3. Events :
4. 1^{st} trim :
5. 2^{nd} trim :

6. 3rd trim:

LABOUR:

Spontaneous: Y/N Induced : Y/N

Indication : _____

Method : Stripping Y/N PGE1 Y/N

PGE2 Y/N ARM Y/N

Synto Y/N

Course of labour stage 1:

Duration	NST	Medication given	MSL	Complications
Normal Y/N Prolonged Y/N	R NR	Analgesics Y/N Antibiotics Y/N Anti HT Y/N Others Y/N	Thick Y/N Moderate Y/N Thin Y/N	Fetal Distress Y/N Others Y/N Specify _____

Course of labour stage 2:

Duration	NST	Medication given	MSL	Complications
Normal Y/N Prolonged Y/N	R NR	Analgesics Y/N Antibiotics Y/N Anti HT Y/N Others Y/N	Thick Y/N Moderate Y/N Thin Y/N	Fetal Distress Y/N Others Y/N Specify _____

Course of labour stage 3:

Duration	Medication given	Complications
Normal Y/N Prolonged Y/N	Analgesics Y/N Antibiotics Y/N Anti HT Y/N Others Y/N	Retained Placenta Y/N PPH Y/N Others Y/N Specify _____

LSCS Y/N

INDICATION	YES/NO
Failed Induction	Y/N
Foetal Distress	Y/N
Deep Transverse Arrest	Y/N
Cephalo Pelvic Disproportion	Y/N
Breech Presentation	Y/N
Previous LSCS	Y/N

Neonate: IP/OP: Sex: B.Wt:

APGAR Score: 1 min 5 min

Resuscitation Y/N

Neonatal Disposition

With mother Y/N

NICU Y/N

HIE I / II / III

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு : cord blood nucleated RBC – a marker of fetal asphyxia

Department of Obstetrics and Gynaecology, KMCH

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் வயது : **பங்கு பெறுபவரின் எண் :**

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. நான் இவ்வாய்வில் தன்னிச்சையாக பங்கேற்கிறேன்.எந்த காரணத்தினாலோ எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக் கொள்ளல்லாம் என்றும் அறிந்து கொண்டேன்.

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்கமாட்டேன்.

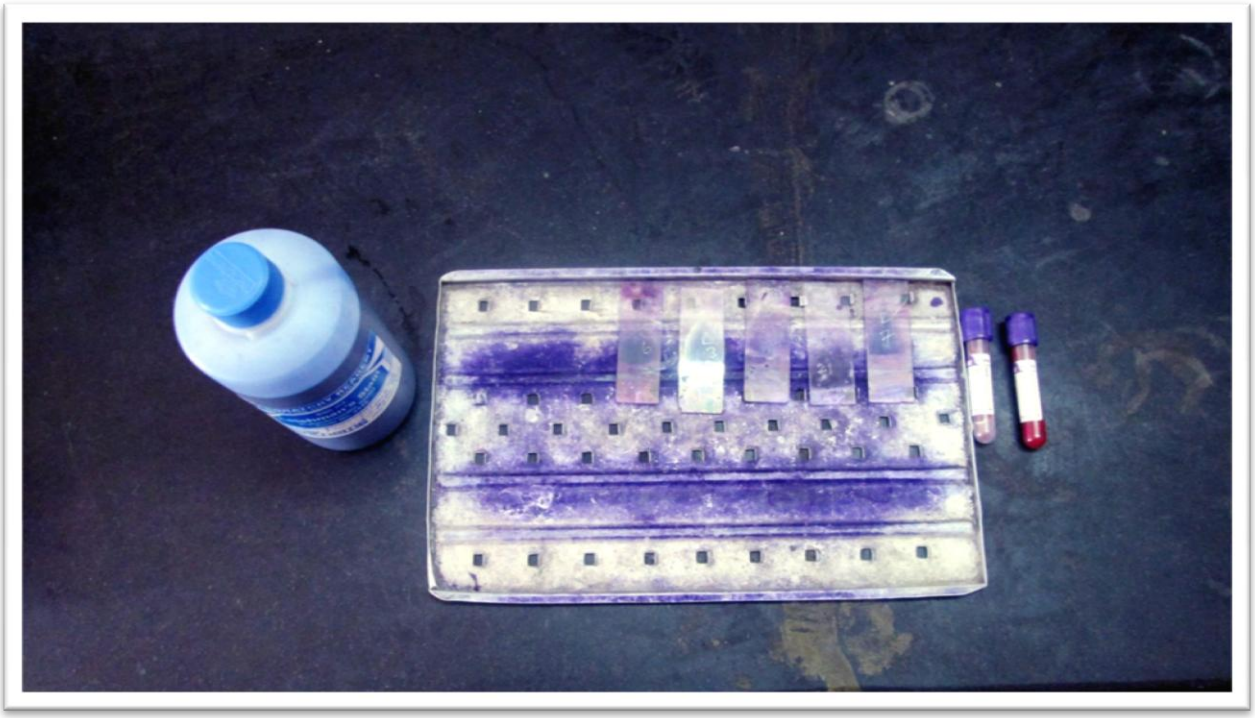
இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன். இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம்
இடம் :
தேதி :
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் :

சாட்சியாளரின் கையொப்பம்
இடம் :
தேதி :

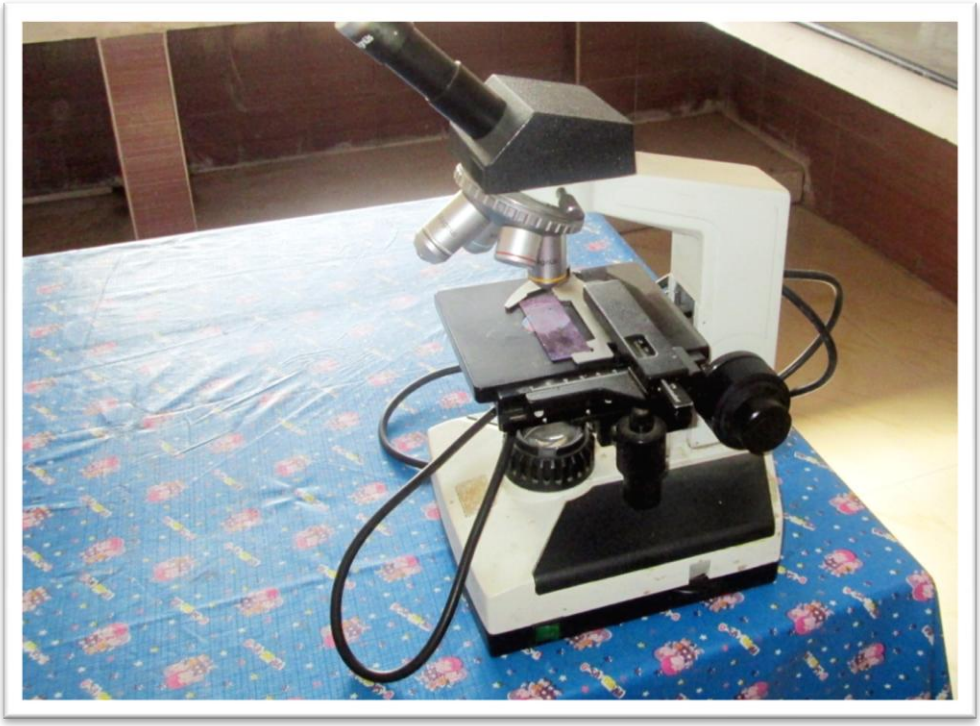
ஆய்வாளரின் கையொப்பம் :
இடம் : தேதி :

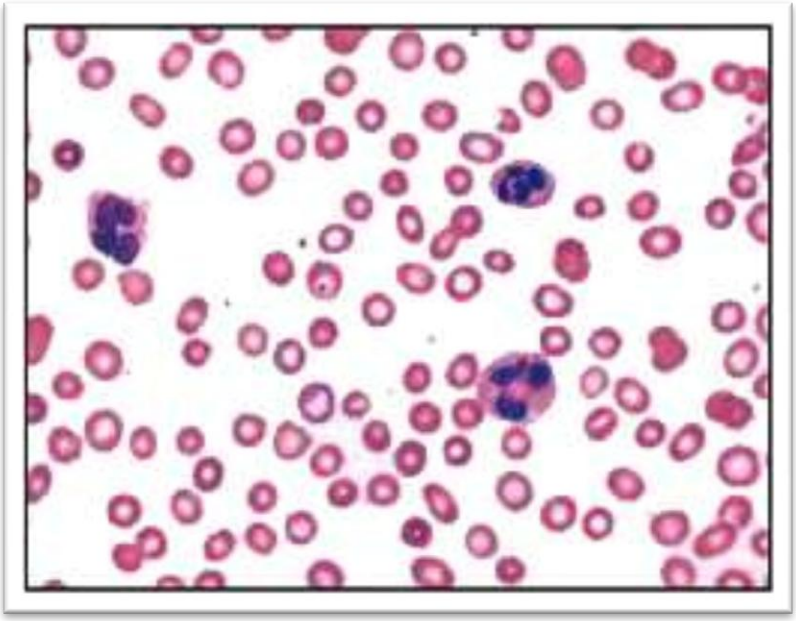
Cord Blood Collection

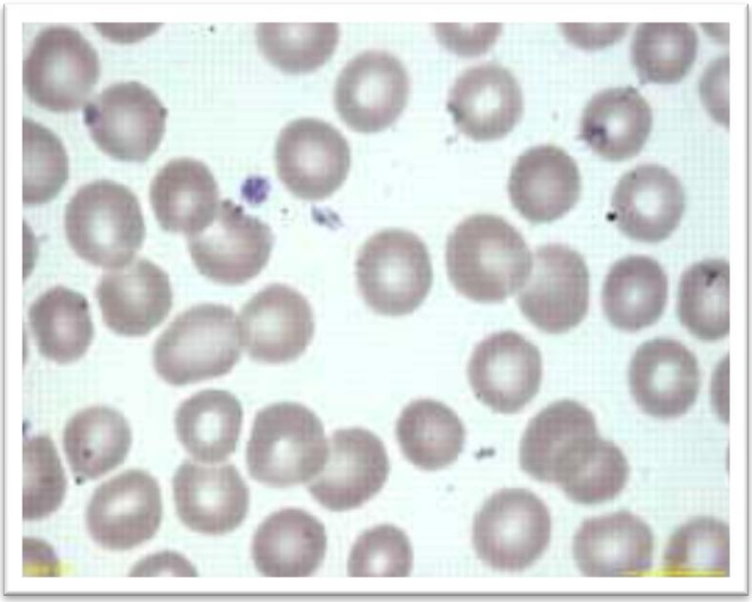


Smear preparation

Microscope with Smear showing NRBCS







HIE-Grade 1

HIE-Grade 2

HIE-Grade 3

Mrs. Mahalakshmi	Age	1318985	Gravida	Parity	Indication	Duration of Labour 1st Stage	Duration of Labour 2nd Stage	Fetal Heart Variability	Colour of Liqour	Apgar 1 min	Apgar 5 min	NRBCs	NICU admission
Mrs.Sathya	24	1319940	1	0	1	0	0	0	0	5	7	2	NO
Mrs.saranya	32	1319680	1	0	6	0	0	0	0	5	8	24	YES
Mrs. Revathy	36	1319938	2	0	1	0	0	0	0	5	7	6	NO
Mrs.Aruna	28	1319943	1	0	4	0	0	0	0	5	8	6	NO
Mrs.Ishwarya	21	1320049	1	0	4	0	2	0	0	4	6	12	YES
Mrs.Gayathri	18	1319936	1	0	2	0	0	1	0	5	7	8	NO
Mrs.Muthulakshmi	22	1320110	2	0	6	0	0	0	0	2	5	18	YES
Mrs.Pachiammal	33	1320111	3	0	6	0	0	0	0	5	7	6	NO
Mrs.Selvarani	28	1320238	2	0	5	0	0	0	0	4	6	12	YES
Mrs.Selvakumari	29	1320220	1	0	6	0	0	1	0	5	7	4	NO
Mrs.bhavani	34	1320237	2	0	6	0	0	0	0	8	9	3	NO
Mrs.Jeeva	36	1320257	3	0	6	0	0	1	1	7	8	8	NO
Mrs.Pavalakodi	19	1320248	1	0	4	0	2	0	0	7	8	6	NO
Mrs.Nrosha	24	1320264	1	0	5	0	0	0	0	5	7	17	YES
Mrs.Sudha	29	1320266	2	0	1	0	0	0	0	7	8	8	NO
Mrs.Shanthi	32	1323044	2	0	6	0	0	0	0	7	8	5	NO
Mrs.Manorama	36	1322696	2	0	6	0	0	0	0	7	8	14	NO
Mrs.Geetha	27	1323223	2	0	2	0	0	1	1	7	8	8	NO
Mrs.Masthana	31	1373260	1	0	4	0	4	0	1	7	8	6	NO
Mrs.Sarala	26	1323424	1	0	1	0	2	0	0	7	8	6	NO
Mrs.Ramyadevi	28	1323804	2	0	1	0	0	0	0	5	7	6	NO
Mrs.Sharvitha	33	1323338	2	0	1	0	2	0	0	7	8	6	NO
Mrs.Revathy	25	1323593	1	0	6	0	0	0	0	5	7	12	NO
Mrs.Sulochana	31	1322667	2	0	5	0	0	0	0	5	7	10	NO
Mrs.Abitala	29	1323342	2	0	1	0	0	0	0	6	8	5	NO
Mrs.Kamala	27	1328735	3	0	4	0	2	0	0	6	8	6	NO
Mrs.Malar	24	132482	1	0	6	0	0	0	0	6	8	6	NO
Mrs.Banu	28	1313455	2	0	2	0	0	1	1	5	7	8	NO
Mrs.Devi	21	1323894	1	0	2	0	0	1	1	5	7	8	NO
Mrs.Muthulakshmi	31	1323857	1	0	1	0	0	0	1	7	8	12	NO
Mrs.Pramila	36	1323932	3	0	2	0	0	1	0	7	8	10	NO
Mrs.Emima	25	1324338	1	0	6	0	0	0	0	2	5	22	YES
Mrs.Subatra	28	1324143	2	0	6	0	0	0	0	7	8	8	NO
Mrs.Gayathri	24	1324289	1	0	2	0	0	1	0	3	6	38	YES
Mrs.Dilshad	22	1324134	1	0	2	0	0	1	0	5	7	8	NO
Mrs.Priya	22	1324672	1	0	6	0	0	0	0	5	7	6	NO
Mrs.Rothibai	19	1324485	1	0	6	0	0	0	0	5	8	6	NO
Mrs.Bhuvaneshwari	21	1324885	1	0	4	0	1	0	0	5	7	5	NO
Mrs.Poongavanam	23	1324349	2	0	2	0	0	0	0	5	7	6	NO
Mrs.Priya	25	1324724	3	0	2	0	1	1	0	5	7	7	NO

Mrs.Subbulakshmi	28	1324423	1	0	5	0	0	0	0	6	8	8	NO
Mrs.Renuka	26	1326749	1	0	6	0	0	0	0	7	8	4	NO
Mrs.Priya	28	1324325	2	0	2	0	0	1	0	7	8	9	NO
Mrs.Indrani	31	1324528	2	0	4	0	0	0	0	7	8	12	YES
Mrs.Gayathri	33	1324907	2	0	4	0	0	0	0	7	8	9	NO
Mrs.Sakunthala	20	1324287	1	0	5	0	0	0	0	7	8	8	YES
Mrs.Bhuvaneshwari	19	1324712	1	0	4	0	0	0	0	7	8	11	NO
Mrs.Saritha	22	1325083	1	0	6	0	0	0	0	7	8	12	NO
Mrs.Umamaheshwari	24	1325035	2	0	5	0	0	0	0	7	8	12	NO
Mrs.Vimala	24	1325305	1	0	2	0	0	1	1	2	5	22	YES
Mrs.Kavitha	22	1325268	1	0	2	0	0	1	0	2	4	28	YES
Mrs.Thulasi	22	1324810	1	0	4	0	0	0	0	7	8	9	NO
Mrs.Varalakshmi	24	1325312	2	0	6	0	0	0	0	7	8	8	NO
Mrs.Malathi	26	1325408	2	0	1	0	0	0	0	7	8	9	NO
Mrs.Mumtaz	25	1324919	1	0	1	0	2	0	0	7	8	8	NO
Mrs.Selvi	26	1324918	1	0	6	0	0	0	0	2	5	28	YES
Mrs.Jaya	26	1325692	2	0	4	0	0	0	0	7	8	12	NO
Mrs.Muthulakshmi	28	1325664	2	0	1	0	0	0	0	7	8	8	NO
Mrs.Anjalai	29	1325683	3	0	1	0	2	0	1	7	8	10	NO
Mrs.Dhanalakshmi	29	1325657	3	0	5	0	0	0	0	7	8	10	NO
Mrs.Mathamani	24	1324909	1	0	4	0	0	0	0	5	7	8	NO
Mrs.Anjalai	24	1327873	1	0	6	0	0	0	0	5	7	8	NO
Mrs.Senthamarai	29	1324893	2	0	1	0	2	0	0	5	7	12	NO
Mrs.Latha	21	1324871	1	0	3	0	1	1	0	5	7	6	NO
Mrs.Mumtaz	30	1324919	3	0	4	0	0	0	0	5	7	9	NO
Mrs.Selvi	29	1324918	2	0	6	0	0	0	0	5	7	12	NO
Mrs.Veerammal	27	1321761	1	0	5	0	0	1	1	5	7	11	NO
Mrs.Rathinam	33	1321764	3	0	1	0	2	0	0	5	7	9	NO
Mrs.Vijayakumari	19	1325681	1	0	1	0	2	0	0	5	7	8	NO
Mrs.Muthulakshmi	26	1325664	1	0	2	0	1	1	0	5	7	6	NO
Mrs.Jaya	29	1325685	2	0	6	0	0	0	0	2	4	32	YES
Mrs.Nirmala	38	1325692	5	0	6	0	0	0	0	5	7	22	YES
Mrs.Vijayalakshmi	34	1325681	1	0	2	0	2	1	1	7	8	6	NO
Mrs.Kasiammal	30	1324080	2	0	1	0	0	0	0	2	5	38	YES
Mrs.Sujatha	27	1325660	1	0	6	0	0	0	0	5	7	8	NO
Mrs.Meenakshi	28	1325644	1	0	5	0	0	0	0	5	7	4	NO
Mrs.Dhanalakshmi	34	1325631	2	0	4	0	0	0	0	5	7	6	NO
Mrs.Anjalai	36	1325683	3	0	1	0	1	0	1	5	7	12	NO
Mrs.Manasarai	30	1324909	1	0	1	0	0	0	0	5	7	4	NO
Mrs.vijaya	32	1324079	1	0	2	0	0	1	1	5	7	4	NO
Mrs.Chinna ponnamal	28	1322364	3	0	4	0	0	0	0	5	7	9	NO
Mrs.Geetha	21	1337470	3	0	5	0	0	0	0	2	5	28	YES
Mrs.Kuppu	30	1417156	2	0	2	0	0	1	0	4	6	7	NO

Mrs.Asha	26	1417158		0	6	0	0	0	0	5	7	8	NO
Mrs.Deviga	29	1417438	4	0	6	0	0	0	0	7	8	12	NO
Mrs.Ezhil Rani	18	1417447	1	0	5	0	0	0	0	7	9	6	NO
Mrs.Anu	22	1417436	3	0	6	0	0	1	0	5	7	10	NO
Mrs.Rani	27	1417428	2	0	6	0	0	0	0	5	7	10	NO
Mrs.Richa	32	1417476	2	0	6	0	0	1	1	5	7	9	NO
Mrs.Swathi	29	1417467	2	0	4	0	2	0	0	5	7	12	NO
Mrs.Anushka	27	1417453	2	0	5	0	0	0	0	5	7	14	NO
Mrs.Divya	24	1417450	1	0	1	0	0	0	0	5	9	8	NO
Mrs.Rekha	25	1417434	1	0	6	0	0	0	0	5	7	9	NO
Mrs.Meenakshi	19	1417351	1	0	6	0	0	0	0	7	9	8	NO
Mrs.jansi	28	1417853	2	0	2	0	0	1	1	2	5	46	YES
Mrs.Soniya	34	1417849	2	0	4	0	4	0	1	5	7	10	NO
Mrs.kasthuri	29	1415691	1	0	1	0	2	0	0	3	5	32	YES
Mrs.selvi	27	1415623	1	0	1	0	0	0	0	5	7	8	NO
Mrs.Jayanthi	22	1415671	1	0	1	0	2	0	0	5	7	7	NO
Mrs.Ammu	19	1418031	1	0	6	0	0	0	0	5	7	7	NO
Mr.kalaivani	29	1418050	1	0	5	0	0	0	0	5	7	4	NO
Mr.Amutha	34	1416045	2	0	5	0	0	0	0	8	9	3	NO
Mrs.Chitra	36	1418146	3	0	4	0	0	0	0	7	8	8	NO
Mrs.Sumathi	19	1418288	1	0	6	0	0	0	0	7	8	6	NO
Mrs.Sasikala	24	1418266	1	0	1	0	2	0	0	5	7	17	YES
Mrs.komathi	29	1415671	2	0	3	0	1	1	0	7	8	8	NO
Mrs.Rajaeshwari	32	1415632	2	0	4	0	0	0	0	7	8	5	NO
Mrs.Sangeetha	36	1418132	2	0	6	0	0	0	0	7	8	14	NO
Mrs.Kamatchi	27	1418473	2	0	5	0	0	1	1	7	8	8	NO
mrs.Anandhi	31	1418514	1	0	1	0	2	0	0	7	8	6	NO
Mrs.Kanagarani	26	1418371	1	0	1	0	2	0	0	7	8	6	NO
Mrs.Dhanalakshmi	28	1418378	2	0	2	0	1	1	0	5	7	6	NO
Mrs.Kavitha	33	1418473	2	0	6	0	0	0	0	7	8	6	NO
Mrs.kanagavalli	25	1418673	1	0	6	0	0	0	0	5	7	12	NO
Mrs.Shree Mathi	31	1418097	2	0	2	0	2	1	1	5	7	10	NO
Mrs.Vijayarani	29	1418285	2	0	1	0	0	0	0	6	8	5	NO
Mrs.mageshwari	27	1418656	3	2	6	0	0	0	0	6	8	6	NO
Mrs.Tamilmathi	24	1418288	1	2	3	0	1	1	0	6	8	6	NO
Mrs.prema	28	1416754	2	2	4	0	0	0	0	5	7	8	NO
Mrs.Ganga	21	1418126	1	1	6	0	0	0	0	5	7	8	NO
Mrs.Varalakshmi	27	1418245	1	5	5	0	0	1	1	5	7	8	NO
Mrs.Janaki	28	1418236	1	5	1	0	2	0	0	5	7	4	NO
Mrs.Anitha	34	1417834	2	5	1	0	2	0	0	5	7	6	NO
Mrs.Vanitha	36	1418934	3	5	2	0	1	1	0	5	7	12	NO
Mrs.Kanmani	30	1414596	1	5	6	0	0	0	0	5	7	4	NO
Mrs.kokila	32	1417645	1	5	6	0	0	0	0	5	7	4	NO

Mrs.Rajathi	28	1416732	3	5	2	0	2	1	1	5	7	9	NO
Mrs.Manimala	21	1417843	3	5	1	0	0	0	0	2	5	28	YES
Mrs.bhuvana	30	1412356	2	5	6	0	0	0	0	4	6	7	NO
Mrs.Nirajana	26	1417642	2	5	5	0	0	0	0	5	7	8	NO
Mrs.komathi	29	1416783	4	5	4	0	0	0	0	7	8	12	NO
Mrs.Savitha	18	1413678	1	5	1	0	1	0	1	7	9	6	NO
Mrs.Geetha Devi	22	1415671	3	5	1	0	0	0	0	5	7	10	NO
Mrs.Hema latha	27	1416732	2	5	2	0	0	1	1	5	7	10	NO
Mrs.Nevetha	32	1416733	2	5	4	0	0	0	0	5	7	9	NO
Mrs. Revathy	29	1416734	2	5	6	0	0	0	0	5	7	12	NO
Mrs.Aruna	27	1416735	2	5	1	0	0	0	0	5	7	14	NO
Mrs.Ishwarya	30	1416736	1	5	4	0	0	0	0	5	7	4	NO
Mrs.Gayathri	32	1416737	1	5	4	0	2	0	0	5	7	4	NO
Mrs.Muthulakshmi	28	1416738	3	5	2	1	0	1	0	5	7	9	NO
Mrs.Pachiammal	21	1416739	3	5	6	1	0	0	0	2	5	28	YES
Mrs.Selvarani	30	1416740	2	5	6	2	0	0	0	4	6	7	NO
Mrs.Selvakumari	26	1416741	2	5	5	2	0	0	0	5	7	8	NO
Mrs.bhavani	29	1416742	4	6	5	2	0	0	0	7	8	12	NO
Mrs.Jeeva	18	1416743	1	6	6	4	0	0	0	7	9	6	NO
Mrs.Pavalakodi	22	1416744	3	6	2	4	0	1	0	5	7	10	NO
Mrs.Nrosha	27	1416745	2	6	4	4	0	0	0	5	7	10	NO
Mrs.Sudha	32	1416746	2	6	4	4	0	0	0	5	7	9	NO
Mrs.Shanthi	29	1416747	2	6	5	4	0	0	0	5	7	12	NO
Mrs.Manorama	27	1416748	2	6	4	4	0	0	0	5	7	14	NO
Mrs.Geetha	24	1416749	1	6	6	4	0	0	0	5	9	8	NO
Mrs.Masthana	25	1416750	1	3	5	4	0	0	0	5	7	9	NO
Mrs.Sarala	19	1416751	1	1	2	4	0	1	1	7	9	8	NO
Mrs.Ramyadevi	28	1416752	2	1	2	4	0	1	0	2	5	46	YES
Mrs.Sharvitha	34	1416753	2	1	4	4	0	0	0	5	7	10	NO
Mrs.Revathy	29	1416754	1	1	6	4	0	0	0	3	5	32	YES
Mrs.Sulochana	27	1416755	1	1	1	4	2	0	0	5	7	8	NO
Mrs.Abitala	22	1416756	1	1	3	4	1	1	0	5	7	7	NO
Mrs.Kamala	36	1416757	2	1	4	4	0	0	0	5	7	6	NO
Mrs.Malar	28	1416758	1	1	6	4	0	0	0	5	8	6	NO
Mrs.Banu	21	1416759	1	1	5	4	0	1	1	4	6	12	YES
Mrs.Devi	18	1416760	1	1	1	6	2	0	0	5	7	8	NO
Mrs.Muthulakshmi	22	1416761	2	1	1	6	2	0	0	2	5	18	YES
Mrs.Pramila	33	1416762	3	1	2	6	1	1	0	5	7	6	NO
Mrs.Emima	28	1416763	2	1	6	6	0	0	0	4	6	12	YES
Mrs.Subatra	29	1416764	1	1	6	6	0	0	0	5	7	4	NO
Mrs.Aayushi	34	1416765	2	1	2	6	2	1	1	8	9	3	NO
Mrs.Aazhikumari	36	1416766	3	1	1	6	2	0	0	7	8	8	NO
Mrs. Aazhimathi	19	1416767	1	1	6	6	0	0	0	7	8	6	NO

Mrs.Aazhinaayagi	24	1416768	1	1	4	6	0	0	0	2	5	22	YES
Mrs. Aazhiyarasi	22	1416769	1	1	1	6	0	0	0	2	4	28	YES
Mrs. Abalya	22	1416770	1	1	1	6	2	0	1	7	8	9	NO
Mrs.Abanisha	24	1416771	2	1	5	6	0	0	0	7	8	8	NO
Mrs.Abarajitha	26	1416772	2	1	4	6	0	0	0	7	8	9	NO
Mrs.Abarana	25	1416773	1	1	6	6	0	0	0	7	8	8	NO
Mrs.Abarika	26	1416774	1	1	1	8	2	0	0	2	5	28	YES
Mrs. Abarna	26	1416775	2	1	3	8	1	1	0	7	8	12	NO
Mrs. Abarnah	28	1416776	2	1	4	8	0	0	0	7	8	8	NO
Mrs. Abarnaya	29	1416777	3	1	6	8	0	0	0	7	8	10	NO
Mrs.Padmasree	29	1416778	3	1	5	8	0	1	1	7	8	10	NO
Mrs.Padmasutha	24	1416779	1	1	1	8	2	0	0	5	7	8	NO
Mrs.Padmavathana	24	1416780	1	1	1	8	2	0	0	5	7	8	NO
Mrs.Padmayani	29	1416781	2	1	5	8	0	0	0	5	7	12	NO
Mrs.Padmini	28	1416782	1	1	4	8	0	0	0	5	7	4	NO
Mrs. Pagalavan	34	1416783	2	1	6	8	0	0	0	5	7	6	NO
Mrs. Pahalavan	36	1416784	3	1	5	8	0	0	0	5	7	12	NO
Mrs.Naamagal	30	1416785	1	1	2	8	0	1	1	5	7	4	NO
Mrs.Naavarasi	32	1416786	1	1	2	8	0	1	0	5	7	4	NO
Mrs.Nadanam	28	1416787	3	1	4	8	0	0	0	5	7	9	NO
Mrs.NadanaMangai	21	1416788	3	1	6	8	0	0	0	2	5	28	YES
Mrs.NadanaMani	30	1416789	2	1	1	8	0	0	0	4	6	7	NO
Mrs.NagaiMuthu	26	1416790	2	1	1	8	2	0	0	5	7	8	NO
Mrs.Nagammal	29	1416791	4	1	6	8	0	0	0	7	8	12	NO
Mrs.Nalayani	18	1416792	1	1	4	8	0	0	0	7	9	6	NO
Mrs.Nallammai	22	1416793	3	1	4	8	2	0	0	5	7	10	NO
Mrs.Nallarasi	27	1416794	2	1	2	10	0	1	0	5	7	10	NO
Mrs.Nallini	32	1416795	2	1	6	10	0	0	0	5	7	9	NO
Mrs.Nambini	27	1416796	1	1	6	10	0	0	0	5	7	11	NO
Mrs.Nangai	33	1416797	3	1	5	10	0	0	0	5	7	9	NO
Mrs.Nanmalar	19	1416798	1	1	6	12	0	1	0	5	7	8	NO
Mrs.Nanmani	26	1416799	1	1	6	12	0	0	0	5	7	6	NO
Mrs.Nanmoli	29	1416800	2	1	6	12	0	1	1	2	4	32	YES
Mrs.Nanmuthu	38	1416801	5	1	4	12	2	0	0	5	7	22	YES
Mrs.Natchelvi	34	1416802	1	1	5	12	0	0	0	7	8	6	NO
Mrs.Natkuna	30	1416803	2	1	1	12	0	0	0	2	5	38	YES
Mrs.Natthamarai	27	1416804	1	1	6	12	0	0	0	5	7	8	NO
Mrs.Neriya	28	1416805	1	1	6	12	0	0	0	5	7	4	NO
Mrs.Neriyaal	34	1416806	2	1	2	12	0	1	1	5	7	6	NO
Mrs.Nila	36	1416807	3	1	1	12	0	0	0	5	7	12	NO
Mrs.Nilani	30	1416808	1	1	1	12	2	0	0	5	7	4	NO
Mrs.Nilavalagi	32	1416809	1	1	6	12	0	0	0	5	7	4	NO
Mrs.Nilavarasi	28	1416810	3	1	4	12	0	0	0	5	7	9	NO

Mrs.Nilavoli	21	1416811	3	1	1	12	0	0	0	2	5	28	YES
Mrs.NiraiMadhi	30	1416812	2	1	1	12	2	0	1	4	6	7	NO
Mrs.Nithila	26	1416813	2	1	5	12	0	0	0	5	7	8	NO
Mrs.Nithilam	29	1416814	4	1	4	12	0	0	0	7	8	12	NO
Mrs.NithilaMan	28	1416815	2	1	6	12	0	0	0	7	8	8	NO
Mrs.Ragini	29	1416816	3	1	1	12	2	0	0	7	8	10	NO
Mrs.Rangammal	29	1416817	3	1	3	12	1	1	0	7	8	10	NO
Mrs.RangaNayagi	24	1416818	1	1	4	12	0	0	0	5	7	8	NO
Mrs.Rangini	24	1416819	1	1	6	12	0	0	0	5	7	8	NO
Mrs.Sembaruthi	29	1416820	2	1	5	12	0	1	1	5	7	12	NO
Mrs.Senbagam	21	1416821	1	1	1	12	2	0	0	5	7	6	NO
Mrs.Sendhen	30	1416822	3	1	1	12	2	0	0	5	7	9	NO
Mrs.Sendalir	29	1416823	2	1	2	12	1	1	0	5	7	12	NO
Mrs.Sentamarai	27	1416824	1	1	6	12	0	0	0	5	7	11	NO
Mrs.Sentamil	33	1416825	3	1	1	12	2	0	0	5	7	9	NO
Mrs.Sevandhi	19	1416826	1	1	6	12	0	0	0	5	7	8	NO
Mrs.Silambu	33	1416827	3	1	4	12	0	0	0	5	7	6	NO
Mrs.Sivakani	28	1416828	2	3	1	12	0	0	0	4	6	12	YES
Mrs.Sivani	29	1416829	1	3	1	12	2	0	1	5	7	4	NO
Mrs.Soodamani	34	1416830	2	3	5	12	0	0	0	8	9	3	NO
Mrs.Sudar	36	1416831	3	3	4	12	0	0	0	7	8	8	NO
Mrs.SudarOli	19	1416832	1	3	6	12	0	0	0	7	8	6	NO
Mrs.Tamarai	24	1416833	1	3	1	12	2	0	0	5	7	17	YES
Mrs.Tamayanti	29	1416834	2	3	3	12	1	1	0	7	8	8	NO
Mrs.Tamilalagi	32	1416835	2	3	4	12	0	0	0	7	8	5	NO
Mrs.TamilArasi	36	1416836	2	3	6	12	0	0	0	7	8	14	NO
Mrs.Tarani	27	1416837	2	3	5	12	0	1	1	7	8	8	NO
Mrs.Teyvanai	31	1416838	1	3	1	12	2	0	0	7	8	6	NO
Mrs.ThanaMalar	26	1416839	1	3	1	12	2	0	0	7	8	6	NO
Mrs.Thangam	28	1416840	2	3	5	12	0	0	0	5	7	6	NO
Mrs.ThavaMalar	33	1416841	2	3	4	12	0	0	0	7	8	6	NO
Mrs.ThenMoli	25	1416842	1	3	6	12	0	0	0	5	7	12	NO
Mrs.Thenral	29	1416843	2	3	5	12	0	0	0	5	7	12	NO
Mrs.ThenSudar	27	1416844	1	3	2	12	0	1	1	5	7	11	NO
Mrs.ThiruMagal	33	1416845	3	3	2	12	0	1	0	5	7	9	NO
Mrs.Thiruvoli	19	1416846	1	3	4	12	0	0	0	5	7	8	NO
Mrs.Thogai	26	1416847	1	3	6	12	0	0	0	5	7	6	NO
Mrs.Thooya	29	1416848	2	3	1	12	0	0	0	2	4	32	YES
Mrs.Thulasi	38	1416849	5	3	1	12	2	0	0	5	7	22	YES
Mrs.DheivaChelvi	34	1416850	1	3	6	12	0	0	0	7	8	6	NO
Mrs.DheivaNayagi	30	1416851	2	3	4	12	0	0	0	2	5	38	YES
Mrs.Logambal	27	1416852	1	3	4	12	2	0	0	5	7	8	NO
Mrs.Loganayaki	28	1416853	1	3	2	12	0	1	0	5	7	4	NO

Mrs.Maalai	34	1416854	2	3	6	12	0	0	0	5	7	6	NO
Mrs.Maalini	36	1416855	3	3	6	12	0	0	0	5	7	12	NO
Mrs.Maargali	30	1416856	1	3	6	12	0	0	0	5	7	4	NO
Mrs.Maari	32	1416857	1	3	2	13	2	1	1	5	7	4	NO
Mrs.Maasila	28	1416858	3	2	1	13	0	0	0	5	7	9	NO
Mrs.Mahilam	21	1416859	3	2	6	13	0	0	0	2	5	28	YES
Mrs.Maina	30	1416860	2	2	3	13	1	1	0	4	6	7	NO
Mrs.MalaiMagal	26	1416861	2	2	4	13	0	0	0	5	7	8	NO
Mrs.Malar	29	1416862	4	2	6	13	0	0	0	7	8	12	NO
Mrs.Malliga	18	1416863	1	2	5	13	0	1	1	7	9	6	NO
Mrs.Mangai	22	1416864	3	2	1	13	2	0	0	5	7	10	NO
Mrs.Mangala	27	1416865	1	2	1	13	2	0	0	5	7	11	NO
Mrs.Manimegalai	33	1416866	3	2	2	13	1	1	0	5	7	9	NO
Mrs.ManiMoli	19	1416867	1	2	6	14	0	0	0	5	7	8	NO
Mrs.Maragadham	26	1416868	1	2	6	14	0	0	0	5	7	6	NO
Mrs.Marudham	29	1416869	2	2	2	14	2	1	1	2	4	32	YES
Mrs.Mayil	38	1416870	5	2	1	14	0	0	0	5	7	22	YES
Mrs.Meena	34	1416871	1	2	6	14	0	0	0	7	8	6	NO
Mrs.Mekala	30	1416872	2	2	5	14	0	0	0	2	5	38	YES
Mrs.Meyyalagi	27	1416873	1	0	4	14	0	0	1	5	7	8	NO
Mrs.Minnal	28	1416874	1	0	2	14	0	1	1	5	7	4	NO
Mrs.Mullai	34	1416875	2	0	2	14	0	1	1	5	7	6	NO
Mrs.Muthalagi	36	1416876	3	0	1	14	0	0	1	5	7	12	NO
Mrs.Muthammal	30	1416877	1	0	4	14	0	0	1	5	7	4	NO
Mrs.Mutholi	32	1416878	1	0	2	14	0	1	1	5	7	4	NO
Mrs.Kaaviya	28	1416879	3	0	4	14	0	0	0	5	7	9	NO
Mrs.Kaveri	21	1416880	3	0	6	14	0	0	1	2	5	28	YES
Mrs.Kadhiroli	30	1416881	2	0	3	14	2	1	1	4	6	7	NO
Mrs.Kalai	26	1416882	2	0	1	14	0	0	1	5	7	8	NO
Mrs.KalaiArasi	29	1416883	4	0	1	14	0	0	0	7	8	12	NO
Mrs.KalaiMagal	28	1416884	2	0	3	14	2	1	1	7	8	8	NO
Mrs.Kanaiyali	29	1416885	3	0	3	14	0	0	0	7	8	10	NO
Mrs.Kanimoli	29	1416886	3	0	6	14	0	0	1	7	8	10	NO
Mrs.Kannagi	24	1416887	1	0	3	14	2	1	1	5	7	8	NO
Mrs.Kannammal	24	1416888	1	0	3	14	1	1	0	5	7	8	NO
Mrs.Katpagam	29	1416889	2	0	3	14	2	1	0	5	7	12	NO
Mrs.Kaviarasi	21	1416890	1	5	2	14	2	1	1	5	7	6	NO
Mrs.Kavimalar	30	1416891	3	5	4	14	0	0	1	5	7	9	NO
Mrs.Kavi Nila	29	1416892	2	5	3	14	1	1	0	5	7	12	NO
Mrs.Kavitha	27	1416893	1	5	3	14	1	1	0	5	7	11	NO
Mrs.Kayal	33	1416894	3	1	3	14	2	1	0	5	7	9	NO
Mrs.Kili	19	1416895	1	1	2	14	2	1	1	5	7	8	NO
Mrs.Kodhai	33	1416896	3	1	2	14	0	1	1	5	7	6	NO

Mrs.KodiMalli	28	1416897	2	1	2	14	0	1	1	4	6	12	YES
Mrs.KodiMullai	29	1416898	1	1	1	14	0	0	1	5	7	4	NO
Mrs.Kohila	34	1416899	2	1	1	14	0	0	1	8	9	3	NO
Mrs.KolaMayil	22	1416900	3	1	1	14	0	0	0	5	7	10	NO
Mrs.KolaVili	27	1416901	2	1	3	14	2	1	1	5	7	10	NO
Mrs.Komagal	32	1416902	2	1	3	14	1	1	0	5	7	9	NO
Mrs.Koormadhi	29	1416903	2	3	3	14	0	0	0	5	7	12	NO
Mrs.Kotravai	27	1416904	2	3	6	14	0	0	1	5	7	14	NO
Mrs.Kulali	30	1416905	1	3	3	14	2	1	1	5	7	4	NO
Mrs.Kumudha	32	1416906	1	3	3	14	1	1	0	5	7	4	NO
Mrs.Kuna	28	1416907	3	4	1	14	0	0	1	5	7	9	NO
Mrs.Kurinji	21	1416908	3	4	4	14	0	0	1	2	5	28	YES
Mrs.Kuvalai	30	1416909	2	4	3	14	0	0	0	4	6	7	NO
Mrs.Kuyili	26	1416910	2	4	6	14	0	0	1	5	7	8	NO
Mrs.OppilaaNangai	29	1416911	4	2	3	14	2	1	1	7	8	12	NO
Mrs.Oviya	18	1416912	1	2	3	14	1	1	0	7	9	6	NO
Mrs.OviyaKodi	22	1416913	3	2	3	16	0	0	0	5	7	10	NO
Mrs.Harini	27	1416914	2	2	6	16	0	0	1	5	7	10	NO
Mrs.Lakshmi	32	1416915	2	2	3	16	2	1	1	5	7	9	NO
Mrs.Manjula	29	1416916	2	2	3	16	1	1	0	5	7	12	NO
Mrs.Nandhini	27	1416917	2	2	1	16	0	0	1	5	7	14	NO

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Ref.No.2212/ME-1/Ethics/2014 Dt:03.04.2014.
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study on cord blood nucleated RBC – A Marker of fetal asphyxia" – For Project Work submitted by Dr.Thenmozhi.G, MS (O&G), PG Student, KMC, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

GOVT. KILPAUK MEDICAL COLLEGE
30 MAY 2014
Kilpauk, Chennai-10.

30/5/14.

CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,Chennai